

Controlling Hospital-Acquired Infection: Focus on the Role of the Environment and New Technologies for Decontamination

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SUMMARY

There is increasing interest in the role of cleaning for managing hospital-acquired infections (HAI). Pathogens such as vancomycin-resistant enterococci (VRE), methicillin-resistant Staphylococcus aureus (MRSA), multiresistant Gram-negative bacilli, norovirus, and Clostridium difficile persist in the health care environment for days. Both detergent- and disinfectant-based

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cleaning can help control these pathogens, although difficulties with measuring cleanliness have compromised the quality of published evidence. Traditional cleaning methods are notoriously inefficient for decontamination, and new approaches have been proposed, including disinfectants, steam, automated dispersal systems, and antimicrobial surfaces. These methods are difficult to evaluate for cost-effectiveness because environmental data are not usually modeled against patient outcome. Recent studies have reported the value of physically removing soil using detergent, compared with more expensive (and toxic) disinfectants. Simple cleaning methods should be evaluated against nonmanual disinfection using standardized sampling and surveillance. Given worldwide concern over escalating antimicrobial resistance, it is clear that more studies on health care decontamination are required. Cleaning schedules should be adapted to reflect clinical risk, location, type of site, and hand touch frequency and should be evaluated for cost versus benefit for both routine and outbreak situations. Forthcoming evidence on the role of antimicrobial surfaces could supplement infection prevention strategies for health care environments, including those targeting multidrug-resistant pathogens.

INTRODUCTION

"here has been much debate over the infection risk to patients from contaminated health care surfaces (1). It is now recognized that the environment may facilitate transmission of several important health care-associated pathogens, including vancomycin-resistant enterococci (VRE), Clostridium difficile, Acinetobacter spp., methicillin-resistant Staphylococcus aureus (MRSA) and norovirus (2–6). These pathogens are frequently shed by patients and staff, whereupon they contaminate surfaces for days and increase the risk of acquisition for other patients (7–14) (Table 1). Environmental screening confirms repeated contamination of items, equipment, and general sites in bed spaces and rooms of colonized or infected patients and often throughout multiple clinical areas in a health care institution (15). Health care workers' hands are liable to touch these contaminated surfaces during patient care, which increases the risk of onward transmission to others (15, 16). Unrecognized environmental reservoirs may also act as a focus for outbreaks or ongoing sporadic transmission (17). Recent studies suggest that the risk of acquiring VRE, MRSA, Acinetobacter spp., Pseudomonas spp., or C. difficile is increased if a new admission is placed in a room previously occupied by a patient known to be colonized or infected with one of these pathogens (18–23). This provides some support for a key environmental role in pathogen transmission. Survival characteristics of individual species or strains on floors and other surfaces could determine the degree of infection risk for patients from inadequately cleaned rooms or bed spaces (7, 24).

Keeping hospitals clean has long been regarded as an esthetic necessity. This has no doubt helped justify the effort and resources involved, since the evidence confirming links between infection risk and contaminated hospitals has only just begun to accumulate (24, 25). In the United Kingdom, cleaning services in the 1990s were an easy target for cost savings in the absence of robust scientific evidence (24, 26–28). The number of housekeeping staff decreased sharply, along with substantial reductions in cleaning hours. Basic cleaning was not thought to be critical for infection control and thus provided an opportunity for cost-cutting (24, 28). From the late 1990s and early 2000s, however, there was a

TABLE 1 Survival times and infectious doses retrieved or extrapolated from published studies a

Organism	Survival time	Infectious dose
Methicillin-resistant	7 days->7 mo	4 CFU
Staphylococcus aureus		
Acinetobacter	3 days->5 mo	250 CFU
Clostridium difficile	>5 mo	5 spores
Vancomycin-resistant Enterococcus	5 days->4 mo	$<10^3$ CFU
Escherichia coli	2 h-16 mo	$10^2 - 10^5$ CFU
Klebsiella	2 h->30 mo	10^2 CFU
Norovirus	8 h–7 days	<20 virions

^a Survival times and infectious doses of a range of pathogens according to, or extrapolated from, original studies, some of which involved animal-based research (2, 7–14).

rapid increase in hospital-acquired MRSA infections in the United Kingdom. This generated much interest in all aspects of pathogen transmission during health care, including pathogen survival and the possibility of environmental reservoirs. Hospital cleaning suddenly became a focus for patients and politicians alike, supported by burgeoning studies confirming the benefits from enhanced cleaning and decontamination during routine and costly outbreak situations (23, 29, 30). Now, both national agencies and local health boards have revisited housekeeping policies to reflect new awareness of the importance of basic hospital hygiene, along with formal monitoring, feedback to cleaners, and surveillance of key environmental pathogens (31, 32). While this recognition is welcome, there are still many controversial issues regarding the place of cleaning for controlling hospital-acquired infection (HAI), compared with, for example, patient screening, isolation, hand hygiene, and antimicrobial stewardship. Current evidence levels can be, and are, challenged over quantity and quality (33-35).

Across the world, the cleaning process itself is subject to debate over frequencies, methods, equipment, benchmarks, monitoring, and standards for surface cleanliness (1, 17). Cleaning policies vary considerably, even within the same health district, and rely heavily upon available resources and political support. While affluent countries debate routine use of nontouch cleaning machines, underdeveloped countries struggle to provide clean water, basic equipment, and cleaning staff. Scientists and clinical microbiologists continue to argue over the value of detergent cleaning (e.g., in the United Kingdom and northern Europe) as opposed to disinfectants (in the United States and Australia) (26, 27, 36, 37). There are governmental targets for HAI rates in some countries, which have helped prioritize infection control, including environmental cleaning practices. In the United States, penalties may be imposed on hospitals that report preventable HAI and poor environmental hygiene. The latter is more usually based upon patient experience and perceptions of cleanliness rather than scientific measurement (38, 39) The package of incentives, financial sanctions, and public reporting requirements no doubt affects operational behaviors and outcomes in hospitals subjected to mandatory inspection.

There are additional issues concerning cleaners themselves, in that many of them receive little or no training for what they are supposed to be doing, and they lack the career progression enjoyed by most other professions (17). There are fewer opportunities for advancement in housekeeping positions, often com-

pounded by language and literacy problems. The status of cleaning personnel, depicted by lower pay scales and basic conditions, does not necessarily reflect the physical cleaning effort and personal risks required to protect patients from hospital pathogens. Janitorial, housekeeping, and domestic staff are regularly confronted by risk of injury, poisoning, or scalding from cleaning equipment and fluids, as well as infection risks from cleaning facilities accommodating patients with transmissible pathogens (17).

This review examines the key evidence for basic cleaning as a major intervention in protecting patients from HAI. Methods for both manual and automated cleaning are presented and discussed, along with the disinfectant debate, range of antimicrobial surfaces, and the need for surface-level standards and routine monitoring. Much of the evidence originates from affluent countries, with United Kingdom cleaning policies chosen to illustrate specific points. Cleaning may be regarded as the most basic infection control activity performed in 21st century hospitals, but it remains of crucial importance, and a great deal more work is required to establish how best to deliver it in a timely and cost-effective manner.

CLEANING AND HAI

While there is still insufficient evidence for the benefits of routine cleaning, it is nearly always mentioned within a package of responses to an outbreak lacking an identified common source (1). A large number of reports include cleaning as an important control component for outbreaks of norovirus, VRE, C. difficile, MRSA, and multidrug-resistant (MDR) Gram-negative bacilli, including Acinetobacter spp. (1, 17, 40). These pathogens thrive in the temperate hospital environment and contaminate numerous sites on surfaces and equipment, including the air (41). Much of the evidence for cleaning is therefore linked to outbreaks, but there are a few studies that focus on the impact of enhanced or alternative cleaning practices on environmental soil in the routine situation (40). Some of these have measured the cleaning effect using standards based on ATP bioluminescence or microbiological screening and modeled this against HAI outcome for patients (42-45) (Fig. 1).

MRSA

Evidence that near-patient surfaces in hospitals could host methicillin-resistant S. aureus (MRSA) was put forward by Boyce et al. in 1997 (46). This study also showed that health care staff could contaminate their gloves by handling or touching sites in close proximity to patients colonized with MRSA. This contrasts with a study published 16 years later, showing that thorough cleaning failed to reduce health care worker gown and glove contamination after caring for patients with MRSA (and multidrug-resistant Acinetobacter spp.) (47). While the risk of health care worker contamination with MRSA remains undetermined therefore, studies have shown that basic cleaning eliminates MRSA from the ward environment, with measured benefit for patients. Over a 14month period, 13 patients acquired MRSA on a dermatology ward despite routine control measures (48). Extensive environmental culturing identified MRSA from the patients' communal shower and a blood pressure cuff, with indistinguishable DNA typing patterns found from both patient and environmental isolates. Cases diminished after enhanced cleaning of shared common areas and changing the blood pressure cuff between patients (48). Another

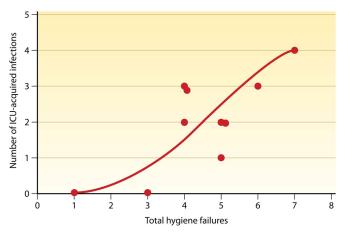


FIG 1 Relationship between environmental bioburden and hospital-acquired infection. This figure shows a relationship between the number of surgical intensive care unit (SICU)-acquired infections and total hygiene fails during a 2-month patient and environmental surveillance study in a Glasgow teaching hospital. Hygiene failures were defined as aerobic colony counts (ACCs) of >2.5 CFU/cm² and/or the presence of *Staphylococcus aureus* on hand touch sites (42).

MRSA outbreak on a urological ward persisted for more than a year, despite implementing all the expected infection control interventions, such as patient isolation and hand hygiene programs (49). After the outbreak strain was recovered from general ward surfaces, the number of cleaning hours was doubled from 60 h to 120 h per week, and there was an immediate reduction in the number of new acquisitions. The authors believed that the extra cleaning was crucial in terminating the outbreak, with cost savings estimated to be at least £28,000 (approximately \$45,000) (49).

Another outbreak of MRSA, this time intermediately resistant to vancomycin, again created problems for infection control staff in an intensive care unit (ICU) setting (50). The outbreak did not resolve until additional measures were introduced, including enhanced cleaning. As several control components were applied together, it was impossible to define the exact effect of either cleaning intervention or barrier precautions. Aside from infection clusters or outbreaks, there is one study examining the effect of targeted cleaning in the routine situation. An enhanced cleaning initiative was introduced into two acute-care surgical wards over two consecutive 6-month periods in a prospective controlled crossover trial (43). The study cleaner worked from Monday to Friday only, prioritizing hand touch sites and clinical equipment for detergent-based cleaning. When the wards received routine cleaning, without any additional attention toward high-risk sites or equipment, nine patients acquired acute infections caused by MRSA, one of whom died and another of whom required surgical intervention. During the enhanced cleaning periods, however, there were just four ward-acquired MRSA infections identified. Based on calculated weekly colonization pressures (MRSA patient-days), statistical analysis predicted 13 new cases of MRSA infection during the enhanced cleaning periods, rather than the four that actually occurred. The study concluded that targeting hand touch sites with detergent wipes could potentially reduce the risk of postoperative MRSA infection, thus saving at least £30,000 (\$51,000) over a 1-year period (43).

Another study conducted on 10 ICUs introduced a new cleaning regimen for rooms previously occupied by patients colonized

with MRSA or VRE (51). The new regimen included a bucket method for soaking cleaning cloths and feedback to cleaners using fluorescent markers. Although the study was quasi-experimental, environmental monitoring showed decreased contamination of room surfaces with MRSA and VRE after initiating the enhanced cleaning (27% versus 45% of cleaned rooms from baseline). Over the same period, patient acquisition of MRSA was reduced by 49% (and that of VRE by 29%) following the augmented cleaning package (P < 0.001 for both) (51).

Two recent studies report decreased rates of MRSA following implementation of a control bundle including targeted screening of patients, environmental sampling, hand hygiene, laboratory methods, and enhanced decontamination of patient rooms. The first used a pulsed xenon UV device (PX-UV) in three American hospitals, with an overall total of 777 beds in the study hospitals (52). Following identification of colonized patients, a 5-day topical clearance protocol was performed, which, along with PX-UV, ultimately reduced the rate of hospital-acquired MRSA acquisition by 56% across the whole health care system after 6 months (P = 0.001) (52).

The second study evaluated the effect of hydrogen peroxide (HP) decontamination alongside patient screening for MRSA in a 300-bed Australian hospital (53). This study ran for 6 years, rather than 6 months, and used a retrospective before-and-after design to assess detergent cleaning versus hydrogen peroxide decontamination of rooms recently occupied by MRSA patients. Targeted environmental screening was performed after room cleaning alongside ongoing surveillance of patient acquisition of MRSA throughout the hospital. Newly identified patients were isolated and placed on contact precautions but were not offered a topical clearance regimen. MRSA was recovered from 25% of rooms following detergent cleaning and from 19% of rooms after exposure to hydrogen peroxide (P < 0.001). There was a 3.5% reduction in the overall proportion of rooms demonstrating persistent MRSA contamination after using hydrogen peroxide (P = 0.08). Over the 6 years, the incidence of MRSA acquisition was reduced from 9.0 to 5.3 per 10,000 patient-days between detergent and disinfectant periods, respectively (P < 0.001).

Both of these studies concluded that enhanced decontamination methods contributed toward decreased MRSA rates, but further work on the individual effects of PX-UV and hydrogen peroxide is warranted (52, 53). As before, the proportional effect from additional screening and other package components in conjunction with introduction of disinfectant or UV light could not be accurately determined.

VRE

It is well known that vancomycin-resistant enterococci (VRE) can survive long term in the hospital environment (7). Multiple cleaning practices fail to remove VRE from a range of sites, despite use of powerful disinfectants (54–57). There are reports showing that surfaces remain contaminated with VRE when cleaning cloths are reused on sequential surfaces, when there is inadequate contact time between a surface and applied disinfectant, and when items or surface are sprayed and wiped over, rather than being actively scrubbed (3, 18, 55, 58). Such persistence is not exclusive to VRE, since other pathogens also survive the cleaning process, but VRE seems to be particularly adept at withstanding repeated attempts at disinfection, including double bleach-based cleaning (54–56).

Current protocols using disinfectants can be effective if near-

patient surfaces, such as bed rails, and frequently touched surfaces, such as door handles, are physically scrubbed at least once daily. There is evidence that more conscientious cleaning can control VRE (3, 51, 57). A sentinel study in 2006 demonstrated the impact of improved cleaning on VRE transmission in a medical ICU, first as a single intervention and then alongside a hand hygiene initiative (58). Targeting cleaning efficiency decreased both surface contamination from VRE and the number of patients acquiring the organism; following this with a hand hygiene program further reduced surface cultures of VRE and patient acquisition to the lowest levels gained. There was also less VRE on health care worker hands (58).

Escalating VRE cases in a Brazilian hospital prompted a range of activities, including emphasis on environmental cleaning, contact precautions, and the introduction of an educational program (59). Improvements in cleaning included use of bleach for bathroom surfaces and 70% alcohol for furniture and patient equipment. The overall package helped prevent dissemination of VRE throughout the hospital, including intensive care, with a decrease in acquisition rate from 1.49 to 0.33 (P < 0.001) (59). Bleachbased terminal cleaning was used for an earlier study to control VRE in a hemato-oncology unit, again as part of an intervention package (57).

Another "bundle" of interventions, including thorough cleaning and surface screening cultures, was implemented in three ICUs by a team in South Korea (60). Clinical and surveillance cultures identified 50 patients with VRE during the outbreak, most of whom (n=46) had vancomycin-resistant *Enterococcus faecium* (VREF). During the first 2 months of the outbreak, PFGE analysis of VREF isolates revealed six main strain types, with related clusters between two of these. Housekeeping staff used 5% sodium hypochlorite to clean all surfaces three times a day. The outbreak finally came to a halt 5 months after implementing the package of interventions, with a reduction in the weekly prevalence rate from 9.1/100 to 0.6/100 patient-days (60).

A comparable study described implementation of a multicomponent package, also based on bleach disinfection, as a response to increasing numbers of patients with VRE (61). Additional cleaning supervisors were appointed to manage the introduction and delivery of a standardized cleaning regimen using a novel product containing detergent and sodium hypochlorite (1,000 ppm). Alcohol-based hand hygiene was encouraged, along with sleeveless aprons instead of long-sleeved gowns and gloves. VRE colonization and/or infection and surface contamination were compared before and after implementation of the infection control package. There was a 24.8% reduction (P = 0.001) in the number of new patients colonized with VRE and a 66.4% reduction (P = 0.012) in environmental contamination, despite a similar proportion of patients already colonized on admission. While VRE bacteremia decreased by over 80% (P < 0.001), the rate of vancomycin-susceptible enterococcal bacteremia did not change during the study (P = 0.54). Susceptible enterococcal infection may well derive from the patient's own endogenous flora, whereas resistant enterococci are more likely to be acquired from persistent surface reservoirs. The "bleach-clean" package encouraged the decline in new VRE acquisition among particularly vulnerable patients alongside an overall reduction in VRE bacteremia rate throughout the hospital (61).

Extreme environmental survival demonstrated by VRE offers an explanation for the increased risk of VRE acquisition for patients placed in a room previously occupied by an individual colonized or infected with VRE (19, 51). The clinical and environmental effects of hydrogen peroxide vapor (HPV) for room disinfection were assessed following discharge of patients with MRSA, *C. difficile*, multiresistant Gram-negative bacilli, and VRE. The risk of acquiring MRSA, *C. difficile*, and multiresistant Gramnegative rods was not significantly reduced after HPV decontamination, but patients admitted into HPV-treated rooms were 80% less likely to acquire VRE (62). This suggests that eradication of persistent reservoirs of VRE may be particularly important for controlling acquisition risk. Cleaning and disinfection should be made a priority for managing VRE and possibly more so than for other hospital pathogens.

C. difficile

The benefits of cleaning for controlling C. difficile are well established (6, 63). The use of chlorine-releasing disinfectants for rooms contaminated with C. difficile reduces the amount of spores in the environment, with additional evidence suggesting that this affects recurrence and transmission of C. difficile-associated infection (CDI) (64). There is particularly good evidence for more concentrated products, especially those releasing higher levels of free chlorine (e.g., 5,000 mg/liter). The benefits of chlorinated products are more obvious in units with high rates of CDI (e.g., those for care of the elderly, stroke rehabilitation, etc.) or if used in conjunction with an outbreak. It should be noted that the overall efficiency of disinfectants for eliminating environmental spores and lowering CDI rates is dependent upon a number of factors, including knowledge and training of cleaning staff, contact time of disinfectants, and overall time allocated to staff for cleaning. Specific strains of C. difficile may also exhibit inherent or acquired properties that make them more resilient to disinfection attempts (64, 65).

A study published in 2007 evaluated additional bleach cleaning in two ICUs following an increase in patients with *C. difficile* (66). The extra cleaning was delivered to all parts of one ICU, including rooms used only by staff. Clinical equipment was cleaned with hypochlorite-containing cloths twice a day. The second unit introduced enhanced bleach cleaning in isolation rooms accommodating patients already infected with *C. difficile*. Both units witnessed a decrease in infection rates over the next few months, which remained at a lower level for at least 2 years after the bleach cleaning program (66).

Increased rates of CDI in three American hospitals prompted a change of disinfectant for terminal room cleaning (67). After discharge of infected patients, all room surfaces from ceiling to floor were wiped over with towels soaked in dilute bleach instead of the usual quaternary ammonium product. The prevalence density of *C. difficile* fell by 48%, with a prolonged and significant reduction in the overall rate of hospital-acquired CDI. Another group implemented 0.55% bleach wipes for daily cleaning of two medical units with a high incidence of *C. difficile* (44). There were 31 patients who acquired *C. difficile* on the wards before the intervention and 4 cases afterwards on these wards over the following year, representing a 7-fold decrease in *C. difficile* cases. There were no other interventions introduced other than targeted cleaning with bleach wipes (44).

A systematic cleaning and disinfection program was assessed by screening frequently touched surfaces for the presence of *C. difficile* in CDI rooms after cleaning (68). Three sequential interven-

tions were introduced over a 21-month period: (i) fluorescent markers placed at key sites for the purposes of monitoring and feedback to cleaners, (ii) use of automated UV equipment for enhanced disinfection, and (iii) support from a designated team responsible for daily assessment of terminally cleaned CDI rooms. The fluorescent marker strategy improved the cleaning quality of frequently touched sites from 47% to 81% (P < 0 0.0001). The number of screened sites positive for C. difficile decreased by 14% (P = 0.024), 48% (P < 0.001), and 89% (P = 0.006) for interventions 1, 2, and 3, respectively, compared with prestudy levels. Positive cultures after disinfection were recovered from two-thirds of CDI rooms before the study began, whereas during periods 1, 2, and 3, the percentages of CDI rooms with positive cultures after disinfection fell by 57%, 35%, and 7%, respectively (68).

More support for the role of cleaning and disinfection in controlling CDI comes from a recent English study (69). The team fitted a statistical breakpoint model against incidence rates of likely hospital-acquired *C. difficile* in a university hospital from 2002 to 2009 and in a district general hospital from 2005 to 2009. The most important infection control interventions during these periods were placed within appropriate categories (antibiotics, cleaning, isolation, and other) for both hospitals and mapped against breakpoints identified by the models. The breakpoints were found to correspond with novel cleaning practices rather than any of the other control interventions. Statistical modeling permitted a means of assessing the impact of different interventions and showed that additional or enhanced cleaning activities were most likely to be responsible for incremental reductions in rates of *C. difficile* at both hospitals (69).

While cleaning and decontamination strategies clearly have an effect on patient acquisition rates, it should be remembered that antimicrobial policies can also be very effective for controlling C. difficile. Severe restrictions on first-line use of cephalosporins and quinolones in a district general hospital reduced acquisition of nosocomial C. difficile by 77% (2.398 to 0.549 cases/1,000 patient beds) (70). The antibiotic policy resulted in an immediate decrease in CDI without any additional infection control interventions. In this study, antibiotic stewardship, not cleaning, was fundamental in controlling C. difficile (70). Beneficial effects of stewardship can be assessed by spatiotemporal modeling, which suggests that protecting the patient from C. difficile acquisition through careful antibiotic choice is more likely to benefit infection control than attempts at curtailing transmission once a patient is symptomatic (71). Faced with a septic patient, however, it is not always possible to restrict antibiotics or choose agents less likely to encourage CDI. Under these circumstances, stringent environmental decontamination should be maintained in order to prevent ongoing transmission.

Acinetobacter

Many studies have emphasized the importance of cleaning in controlling outbreaks of *Acinetobacter* spp., particularly those caused by multiresistant strains in critical care units (4, 72, 73). One study describes an outbreak due to multiresistant strains of *A. baumannii* involving more than 30 patients in two ICUs (4). Epidemic strains were identified from environmental reservoirs throughout both of the affected ICUs, which ultimately required complete closure for terminal disinfection in order to bring the outbreak to an end (4). Another study reported a prolonged outbreak in a neurosurgical ICU, which prompted ongoing environmental

sampling in order to identify any persistent reservoirs (74). The epidemic strain was frequently isolated from hand touch sites beside patients, with a clear association demonstrated between the levels of surface contamination and new patient acquisition. The authors stated that comprehensive cleaning is fundamental for controlling *Acinetobacter* outbreaks in ICU settings, although the most appropriate cleaning practices in the routine situation remain ill-defined (74).

One further study involving spread of a multiresistant *A. baumannii* strain in a critical care unit also provides environmental sampling data during an outbreak affecting over 60 patients (75). Once again, there appeared to be a relationship between the number of positive environmental cultures and new patient cases. The authors stated that systematic screening allowed them to target cleaning resources in order to gain control of the outbreak (75).

An investigation following a sudden increase in the number of children acquiring *Acinetobacter* on a pediatric burn ward identified the role of frequently handled clinical equipment as an outbreak reservoir (76). The outbreak occurred after it was decided to install computers beside every child's bed. Environmental screening identified the organism on several surfaces in the children's rooms, including the plastic covers on top of the computer keyboards. Until the outbreak occurred, there had been no recommendation for including bedside computers and their components in the routine cleaning specification. Targeted infection control measures were introduced, which included decontamination of the plastic covers and mandatory glove use for staff before handling the computers. These simple measures were effective in stopping the outbreak (76).

A 3-year prospective study took place in intensive and coronary care units in order to evaluate a bundle of interventions aimed at reducing long-term drug-resistant *Acinetobacter* (77). The interventions included a hand hygiene program, patient surveillance, barrier precautions, contact isolation, cohorting affected patients, and intensive cleaning with sodium hypochlorite (1:100) (77). The rate of *A. baumannii* colonization and/or infection was 3.6 cases per 1,000 patient-days before the interventions were introduced, with the rate then decreasing by 66% to 1.2 cases per 1,000 patient-days (P < 0.001) by the end of the first year. The rate was further reduced by 76% to 0.85 cases per 1,000 patient-days (P < 0.001) 2 years later (77).

Another outbreak of *Acinetobacter* in an ICU affected 18 patients and was traced to a sink in one of the patient rooms (78). Identification of the sink trap as the reservoir suggested that the whole of the horizontal drainage system could be potentially contaminated. Application of a bleaching protocol eradicated the reservoir and curtailed further acquisition of MDR *A. baumannii*. However, there were additional infection control measures introduced at the same time, which included contact isolation for every patient identified with MDR *A. baumannii*, hand hygiene training, additional nurse teaching, use of an alcohol hand gel, and direct observation of cleaning in the ICU (78). Once again, it is impossible to extricate the contribution of reservoir decontamination when several interventions were initiated simultaneously as part of an outbreak control package.

One further study provides evidence to support the importance of cleaning in controlling outbreaks of *Acinetobacter* (79). As with most of the studies described, this outbreak also occurred in an ICU, and an extremely resistant outbreak strain resisted carbapenem antibiotics. Carbapenem-resistant *A. baumannii* was grown

from multiple environmental samples during the outbreak, including a mattress, a vital signs monitor, near-patient horizontal surfaces, computer components, and a glucometer. After failure of thorough cleaning attempts with detergent and alcohol wipes, a commercial oxidizing disinfectant (Virkon S [50% potassium peroxomonosulfate, 15% sodium alkyl benzene sulfonate, and 5% sulfamic acid]) was selected for enhanced cleaning. The introduction of Virkon-based cleaning rapidly brought the outbreak to a close. The authors were uneasy about the temporal association, because epidemics can resolve of their own accord. Furthermore, they did not audit cleaning effectiveness, hand hygiene compliance, antimicrobial consumption, or other potentially confounding factors. However, the sudden and sustained decrease in the number of cases of infection with a carbapenem-resistant A. baumannii strain after implementing use of a powerful new disinfectant is compelling (79).

It appears that even stringent manual cleaning with disinfection does not necessarily eliminate *Acinetobacter* completely from the environment. The reasons for this are unknown but probably include poor cleaning practices, missing high-risk sites, overwhelming bioburden, and tolerance to, or misuse of, disinfectants (80, 81). In another study, surfaces in rooms occupied by patients colonized with *A. baumannii* remained contaminated with the organism despite disinfectant-based cleaning (81). This study also reported contamination of rooms accommodating patients not previously shown to have any recent cultures of *A. baumannii*, suggesting long-term persistence in the near-patient environment. There was a significant reduction in *Acinetobacter* contamination following disinfection, but over half the rooms that were positive prior to cleaning still harbored the organism on a range of surfaces after cleaning (81).

Multidrug-Resistant Gram-Negative Bacilli

While the role of cleaning in controlling *Acinetobacter* outbreaks is now accepted, the same cannot be said in relation to outbreaks of multidrug-resistant (MDR) Gram-negative bacilli. As for any outbreak, enhanced cleaning usually comes as part of an overall bundle of activities in reaction to cross-infection incidents (1). There are, however, plenty of reports detailing coliforms associated with discrete items of equipment, specific environmental reservoirs, or a particular product or practice during outbreak investigations (24). Finding a single reservoir and eradicating it usually stops an outbreak, and a positive outcome would naturally encourage publication (82–85). Terminating an outbreak caused by single-source contamination is much easier to achieve than implementing a widespread cleaning regimen that has to cover a multitude of diverse items and surfaces.

Away from the outbreak situation, it has long been assumed that Gram-negative bacteria survive poorly on surfaces. This means that any environmental contribution toward HAI by this group of organisms has not been widely investigated. Recent work has challenged this, and there is a growing consensus that environmental cleanliness could be just as important for controlling transmission of MDR coliforms as it is for MRSA and other organisms (86, 87). This is supported by studies showing that *Escherichia coli* and *Klebsiella* spp. may survive desiccation for more than a year and *Serratia marcescens* for several months (7). There are additional reports demonstrating persistence of MDR coliforms throughout a variety of health care environments, with some evidence that MDR *Klebsiella* is recovered from surfaces

more often than MDR *E. coli* (15, 88–91). One recent study screened the near-patient environment beside patients previously identified with carbapenem-resistant *Enterobacteriaceae* (CRE) and found that about 25% of the sites tested were contaminated, presumably by the patients' own organisms (90). This study also demonstrated that both timing of sampling and local cleaning strategies could affect data on the frequency of environmental contamination by CRE. This is no doubt true for other environmental pathogens.

Other than sampling and cleaning practices, it is possible that a lack of evidence for viable MDR coliforms and corresponding infection risk posed by hospital surfaces is due to insensitive screening methods (90, 92). A targeted recovery strategy was used to sample frequently touched surfaces situated beside patients colonized by MDR coliforms (light switch, bed rail, bedside locker, and mattress cover) and two sites in nearby bathrooms shared by patients (shower handrails and sink faucets) (92). Environmental screening next to one of these patients recovered MDR Klebsiella pneumoniae from four of six sites sampled, all of which were indistinguishable from the strain obtained from the same patient's urine. The sites contaminated with the MDR strain were either beside this patient or from the adjacent communal bathroom. Given the low recovery rates, limited detection, and relatively short survival times (1.5 to 2 h), isolating even small numbers of MDR coliforms suggested a relatively high initial burden on surfaces. Contamination probably occurred within a short time before sampling (92).

Hospital sinks represent one of the most frequently implicated reservoirs for MDR Gram-negative bacilli, including MDR coliforms (93, 94). K. pneumoniae strains demonstrating prolonged survival within plumbing components are also more likely to harbor extended-spectrum β-lactamases (95). Persistent reservoirs of resistant K. pneumoniae were detected from multiple sites associated with a contaminated sink in a large Scottish hospital (83). More recently, four patients in a neurosurgical ICU acquired MDR K. pneumoniae thought to have originated from another contaminated sink during a 7-month period (85). Removal and replacement of the sink and related pipes and upgrading the practices for sink usage and decontamination brought the outbreak to an end. A protracted clonal outbreak of multiresistant IMP-8producing Klebsiella oxytoca in a Spanish ICU was finally terminated by removing sinks, drain and trap components, and even the horizontal system connecting all suspected sinks (96). If the usual control measures fail to terminate an outbreak, then alternative and/or unusual reservoirs should always be considered, particularly when preliminary environmental screening is nega-

Another outbreak of MDR *Klebsiella* was linked with tipping patient fluids down the nearest available sink rather than taking clinical waste to the designated sluice further away (97). A recent audit of sinks in ICU rooms suggested that lower rates of sink contamination are significantly associated with daily bleach disinfection, as well as restricting sinks for hand washing only and not routine disposal of fluid waste from patients (94).

Yet another outbreak of resistant *K. pneumoniae* highlights the risks of reusing disposable equipment (84). This outbreak involved neonates, most of whom were infected just after birth or within a few days of hospitalization. Cases occurred among those babies receiving mucous aspiration due to respiratory distress. Although a new aspiration tube was used for each separate baby, it

was cleaned only by rinsing in a bowl of tap water between aspiration episodes for the same baby. The bowl was not routinely cleaned, and the water was left unchanged between babies. Not surprisingly, the water was found to be contaminated with the same resistant *K. pneumoniae* strain (84).

The lack of evidence for benefit from general surface cleaning alone for MDR Gram-negative organisms, even as a response to an outbreak, is well recognized (98). There is a recent report emphasizing additional cleaning following recovery of a carbapenemase-producing K. pneumoniae from patients in a United Kingdom hospital (99). Chlorine-based cleaning was implemented throughout the ward, including patient-related items. Additional cleaning was only one component of the overall infection control strategy, however, along with a urinary catheter care bundle, tagging of patient notes, improved hand hygiene, and contact precautions for all cases (99). Another report describes an educational intervention to improve environmental cleaning and hand hygiene in an 11-bed gastrointestinal surgical ICU (100). There may well have been an underlying outbreak at the start of this initiative, since a high proportion of patients appeared to be already colonized. Following the introduction of terminal cleaning with glutaraldehyde, single-use equipment, barrier precautions, and hand hygiene improvements, the number of patients colonized with MDR Enterobacteriaceae decreased from 70% to 40%, attributed to the overall interventional package (100).

Pseudomonas and Stenotrophomonas spp.

Despite lack of evidence for defined transmission pathways, there are studies suggesting that water sources provide a reservoir for Pseudomonas and Stenotrophomonas spp. in the health care environment (101). These opportunistic organisms pose a risk of colonization and infection for particularly vulnerable patients. One previous study showed that Pseudomonas aeruginosa may be transmitted from contaminated sinks to hands during hand washing (102). While survival on dry surfaces may only be transient, persistent reservoirs of these organisms can be traced to biofilm adherent to surfaces on sinks, sink traps, pipes, water lines, and hospital drains (103, 104). Biofilm is made up of a multifaceted matrix of living organisms, which contaminates internal plumbing and provides a long-term reservoir for water-associated organisms, including pathogens. The biofilm structure itself is resilient and situated on multiple surfaces inside traps, pipes, and internal water filters. Bacteria present within biofilm are more likely to be able to withstand chlorine-containing and other types of disinfectants. They are also likely to demonstrate an increased capacity for antimicrobial resistance (95, 105).

Various outbreak investigations have shown that recovery of *Pseudomonas* and *Stenotrophomonas maltophilia* from water sources and adjacent surfaces can be linked with indistinguishable strains cultured from patient specimens (106–108). An outbreak of *Burkholderia cepacia* on a pediatric unit was traced to sinks and was thought to be associated with the presence of aerator filters fitted to the taps (109). Faucet aerators have also been implicated in an outbreak of *S. maltophilia* in a surgical ICU, with pulsed-field gel electrophoresis (PFGE) illustrating indistinguishable strains from patients and aerators (106). For this reason, aerators should be replaced with flow straighteners in health care premises.

Exposing biofilm to chlorine-containing products is the usual reaction to disinfection attempts, but even prolonged irrigation fails to remove all adherent biofilm. Reliable control requires

stringent and repeated cleaning strategies, aimed at physical disruption of the biofilm lining the internal surfaces of affected water systems (108, 110). These are often right beside patients in the clinical environment and difficult, or even impossible, to access. Infection control initiatives require close collaboration between structural facilities, clinical, and housekeeping staffs in order to safely replace components or remove persistent biofilm. Total eradication is rarely achieved, but regular inspection and repeated cleaning followed by chlorine-based or similar disinfection will hinder further cases. Long-term control of *Pseudomonas* and *Stenotrophomonas* is dependent upon integration of an effective cleaning strategy into a targeted maintenance program (17, 101).

Norovirus

While the environmental role in the transmission of norovirus is difficult to prove, the most convincing evidence comes from outbreaks where groups in a common setting with no known direct contact have been sequentially affected. The best examples of these come from outbreaks occurring outside hospitals. One report involves a single aircraft on which a single passenger vomited during a long-haul flight (111). Over the next 6 days, flight attendants working on the aircraft in multiple flight sectors developed gastroenteritis. Analysis of specimens from these aircrew attendants demonstrated an unusual norovirus genotype. The only possible exposure was working in the cabin environment, since there were no other opportunities for person-to-person transmission (111).

Another study describes an outbreak linked to a public concert hall (112). More than 300 people developed gastroenteritis during a five-day period after a concert attendee vomited in the hall. The highest risk occurred among people seated closest to the seat belonging to the original attendee. Similar events were recorded on a cruise ship, where six consecutive cruises were affected (113). While crew members may have carried the virus between cruises, it is highly likely that the linked series of outbreaks was due to environmental persistence of infectious norovirus. These incidents suggest that without scrupulous cleaning following a single incident, outbreaks will commence, escalate, or even resume.

Outbreaks of norovirus can be particularly ferocious in closed or semiclosed communities, such as transport vehicles and a variety of public venues (114, 115). Sudden and widespread outbreaks can escalate without warning in nursing and residential homes, schools, hotels, and prisons (114, 116-118). CDC reported an outbreak of norovirus in a primary school that affected over 100 staff members and pupils (117). The investigation following this outbreak identified person-to-person contact as a major factor in viral transmission, but there was evidence that the environment was also implicated. Despite intensive cleaning with bleach soon after notification, norovirus was recovered from computer components in a frequently used classroom the next day. The environmental strain was indistinguishable from that retrieved from symptomatic patients. Public health staff excluded symptomatic cases from the school, advised hand hygiene improvements, and organized additional 1:50 bleach cleaning of environmental sites that might have been overlooked during the original disinfection strategy (117).

The role of cleaning in the control of norovirus outbreaks in hospitals and other health care facilities is unquestioned (5, 116). Indistinguishable genotypes of norovirus from ward surfaces and patients have been reported, with viable virus apparently surviving enhanced cleaning (119). One recent study identified norovi-

rus reservoirs from expected sites near bathroom showers and toilets, but ward-based screening also demonstrated viral contamination of near-patient sites and a wide range of clinical equipment, including blood pressure and pulse oximeter machines, thermometers, notes trolleys, and even soap and alcohol gel containers. Persistent viral reservoirs place new admissions at continued risk of norovirus acquisition. Indeed, overloaded health care facilities may experience prolonged outbreaks, especially if confronted with a higher throughput of patients lacking prior exposure (119).

All cleaning specifications, particularly regarding toilets and bathrooms, should use chlorine-based disinfectants at an appropriate concentration for norovirus outbreaks. Detergent-based cleaning is not sufficient to eliminate norovirus from the environment (120). A recent in vitro study measured residual contamination of surfaces with norovirus after detergent cleaning with or without a disinfectant (121). The authors concluded that cleaning with liquid soap followed by a 1,000-ppm chlorine wipe generally produced the lowest level of persistent contamination. The infectivity index of norovirus, however, meant that even the low levels achieved after a two-tier approach would still represent a risk for hand contact transmission. The authors suggested lengthening the contact time between chlorinated disinfectant and contaminated surfaces to a minimum of 5 min, since this reduced residual levels of virus to less than those capable of causing infection (121). Translating the results from this study to the clinical environment poses a challenge, since leaving disinfectants on surfaces for even 5 min in a busy ward may not be practical.

MANUAL CLEANING: PROCESS AND EQUIPMENT

Routine Cleaning Practices

In hospitals, environmental surfaces are routinely cleaned, or cleaned and disinfected, according to predetermined cleaning policies (e.g., hourly, daily, twice weekly, etc.) or when surfaces appear visibly dirty, if there are spillages, and always after patient discharge (31, 122). The type and frequency of routine cleaning depend upon clinical risk, patient turnover, intensity of people traffic, and surface characteristics. Frequent and stringent cleaning specifications are applied to areas within operating theaters, intensive care units, transplant wards, and so-called "clean" rooms, where sterile medications are decanted and/or processed. Hospital kitchens, restaurants, and cafes also require targeted frequent cleaning, as do the laboratories and staff on-call rooms. Less comprehensive cleaning regimens are carried out for corridors and stairwells, offices and waiting rooms, and selected outpatient, storage, general purpose, and entrance areas.

All hospitals should provide a written specification of cleaning services and their delivery for all areas of the hospital, whether provided by in-house or externally contracted staff (31, 122, 123). These should be reviewed on a regular basis by cleaning supervisors, hospital managers, and structural facilities and infection control personnel. Recent recommendations on innovation and research in infection control support the opportunity for hospitals to test new cleaning and decontamination technologies and publish their findings (124).

In the United Kingdom, routine cleaning is performed manually, with basic equipment, including buckets, mops, brushes, brooms, wipes, and cloths (31, 122). Electrical equipment includes vacuum cleaners, floor polishers, and scrubbing machines.

Surfaces fall into two general categories: critical and noncritical surfaces. The latter encompass sites such as floors, furniture, soft furnishings (including curtains), doors, wall fixtures, ledges and shelves, radiators, ceilings and walls, grilles and other ventilation components, cupboards, etc. Critical surfaces include those that are frequently touched or handled, such as handles, buttons, switches, computer keyboards, and bed controls, and noninvasive clinical equipment, such as electrocardiogram (ECG) machines, blood pressure cuffs, patient hoists, stethoscopes, and intravenous drip stands.

Noncritical Surfaces

Neutral detergent is used to lift soil, using disposable or reusable materials. Over 80% of the bacterial load on hospital floors can be removed by detergent-based cleaning only (125). Water used for mop rinsing usually becomes increasingly contaminated during this process, especially if used repeatedly without changing or if surfaces are heavily soiled and/or have not been cleaned within the previous 24 h. The water then serves as a medium for spreading microbes around the environment. It should be routinely discarded in favor of fresh detergent solutions between bed spaces or every 15 min, whichever is sooner (122). Disinfectants can be used for floors in high-risk clinical areas, although there is no evidence that any microbial reduction persists for substantially longer periods than that achieved by detergent alone (26, 27).

Mop heads may be disposable, with the length of time and/or areas of use specified; if not, they are employed for a particular duty, e.g., operating theater, before being bagged and sent for decontamination, usually on a daily basis (122). Failure to adequately decontaminate reusable materials permits survival of microbes, including spores, which may then contaminate the next surface to be cleaned. This may occur despite use of disinfectants, since certain organisms can resist the effect of specific chemical agents either naturally, through acquired resistance, or protected by biofilm (126–128).

Both detergent and disinfectant wipes and cloths can be used to wipe over noncritical surfaces on a routine basis, with disposable products obviating the need for decontamination (122). Cleaning staff require education on which product can be used for which surface and how long a wipe or cloth should be used before disposal. As a general guide, one wipe or cloth can be used for noncritical surfaces in one room or bed space, not including bathroom areas. Cleaning materials for the latter should always be kept separate from those used for other ward surfaces (122). Disposable wipes are quick and easy to use but may leave excess moisture or residues on surfaces, which can attract additional soil and ultimately spoil the finished appearance. They may also be expensive and cause allergic reactions among housekeepers, with or without protective clothing, including gloves.

Automated assistance includes vacuum and steam cleaners as well as floor scrubbers and polishers. Use of a vacuum cleaner before wet mopping reduces overall soil, which may otherwise be spread around during the mopping process (24). Scrubbing machines achieve a high standard of cleanliness for floors and are often used for cleaning operating theaters on a routine basis (125). There is a longer-term beneficial microbiological effect seen after using these machines, but they tend to be cumbersome as well as labor-intensive (125).

Critical Surfaces

Frequently touched items such as telephones, handles, taps, light switches, levers, knobs, buttons, keyboards, push plates, toys, etc., are found in most health care institutions. Repeated handling increases the risk of contamination by pathogens, which then leads to hand-based transmission. These items are likely to benefit from enhanced cleaning, including disinfection (123, 129). High-touch sites or surfaces can be identified through direct observation or environmental screening using fluorescent or other markers (130).

A study performed in 1999 described the inoculation of a telephone handle in the middle of a neonatal ICU using fragments of cauliflower mosaic virus. Over the ensuing week, the study team tracked dispersal of the viral pieces around the unit between hand touch sites (131). Before inoculating the telephone, over 30 sites for sampling were chosen in each of six patient rooms according to the risk of direct or indirect transmission of pathogens. These sites included equipment buttons, handles, computers, patient charts, and hand lotion dispensers. Over half (58%) of the sites screened in the room containing the inoculated telephone were persistently contaminated with the DNA marker. The number of sites positive for viral markers peaked at 8 h (78%) before declining to 23% 1 week later. Around 18% of sites were positive in the remaining five rooms throughout the week, with a similar decline. The most commonly contaminated sites in all six rooms were personnel hands, computers, blood gas analyzers, door and telephone handles, control buttons and knobs, patient monitors, and medical charts (131). Such data specifically highlight the areas that would benefit from more frequent cleaning or disinfection. The recognition of high-risk sites for potential pathogen transmission utilizes principles employed by the food industry, whereby a monitoring framework is constructed specifically to prevent contamination during food production (132). This framework is based on a hazard analysis critical control point (HACCP) system and aims to eliminate risk through a variety of integrated control strategies

Near-patient hand touch sites constitute the bulk of critical surfaces in a ward. Routine decontamination is usually included within institutional cleaning policies, including designated tasks for a range of staff. This can vary between occupied and nonoccupied beds, electrical and nonelectrical items, and clinical and nonclinical equipment, all of which illustrates cleaning complexities and the potential for fragmented responsibility between house-keeping, nursing, and other clinical staff. Daily attention with detergent wipes may be sufficient to control bioburden on an acutecare ward, but high-risk sites in intensive care units may require more frequent attention (133). Two studies have clearly shown how MRSA rapidly recontaminates high-touch sites in the ICU setting after cleaning (134, 135).

Clinical Equipment

Given the range and types of clinical equipment available in today's hospitals, it is beyond the scope of this review to describe and compare decontamination strategies for all items that might be found on a ward. There is, however, an important decontamination principle related to clinical equipment that applies to any item used for patients excluded from routine domestic specification (136, 137). All clinical equipment should be cleaned and/or decontaminated before and after use for all patients regardless of how often it is used, where it is used, or what it is. An item intended for patient use should be inspected carefully before it is employed, and if prior cleaning is not evident by either notification or obvious soiling, then it should be immediately cleaned according to local policies. Many hospitals now ask staff to flag specific pieces of equipment to show that they were appropriately cleaned and/or decontaminated after use. This is especially important for items such as commodes and other nondisposable apparatus used for toileting and therefore at high risk of contamination. There are other utensils that might come into contact with blood and/or body fluids, e.g., pulse oximeters, thermometers, blood sugar test kits, saturation probes, etc., and these should also be subjected to stringent cleaning and disinfection before and after use. Doctors' stethoscopes have long been the subject of cleaning audits and remain a likely source of contamination for a range of microbial flora (138). Wiping with alcohol is effective for decontaminating stethoscopes, but it appears that even this simple procedure is abandoned, ignored, or forgotten when staff are overworked. Indeed, all hygienic practices are consistently challenged on a busy ward (139).

A recent unannounced audit conducted on an acute-care ward discovered various amounts of organic soil on many items of clinical equipment (136). The authors used ATP bioluminescence to measure soil and found that 84% of sampled items exceeded the benchmark value provided by the device manufacturer. The audit identified several items of equipment on the ward that lacked any designated cleaning responsibility, and these tended to show higher levels of contamination. The results are comparable with data from a previous audit, which reported a pooled mean of 86.8% contamination of equipment, although this study used microbiological sampling methods rather than ATP bioluminescence (137).

In many hospitals nowadays, nurses have adopted or taken on a range of duties originally performed by doctors, e.g., intravenous line insertion, prescribing, and catheter manipulation. Given these specialist tasks, it is understandable that basic cleaning has been overlooked following the current shift in professional responsibilities (136, 140). Cleaning duties do not necessarily represent an appropriate use of time for highly trained nurses (141). Housekeeping staff are expected to comply with policies that often lack detailed guidance for each and every item found on a ward. Furthermore, they are not usually trained to decontaminate electrical items or clinical equipment (31, 122). Taking these changes together, there is a risk that frequently used equipment and socalled forgotten sites will accumulate soil, including opportunistic pathogens. Since only a few spores of C. difficile or CFU of S. aureus can initiate infection at a vulnerable site, persistent contamination of soiled items provides a continued risk to patients (Table 1) (2, 10). It is likely that numerous items of clinical equipment in health care settings receive only sporadic cleaning attention or, perhaps, none at all. Cleaning and decontamination responsibilities for all staff, including medical staff, should be regularly reviewed, along with appropriate and repeated training programs (136, 140).

Terminal (Deep) Cleaning

Terminal or deep cleaning is performed following patient discharge (122). If the patient was known to be colonized or infected with a specific pathogen, then the cleaning regimen is usually augmented with disinfectant at a specified strength depending upon

the pathogen. Methods vary, but a terminal clean usually includes initial removal of all detachable objects from the room, including bedding, screens, and/or curtains. Lighting and ventilation components on the ceiling are dusted or wiped over, followed by curtain rails and the upper surfaces of highly placed fixtures and fittings. All other sites and surfaces are then cleaned downward to floor level. Items and equipment removed from the room are wiped over with detergent cloths, alcohol wipes, or disinfectant before being replaced.

A terminal clean also implies removal of curtains, drapes, and screens for laundering or cleaning; fixed blinds may be wiped over in situ. While housekeeping staff are assigned to deliver terminal cleaning of bed spaces and patient rooms, nurses, nursing auxiliaries, and clinical support workers usually have responsibility for clinical equipment and electrical appliances, including beds. This division of labor creates confusion over who cleans what, unless clear contractual obligations are provided (136, 140). A flexible approach in terms of responsibility between nurses and housekeeping services must be adopted to ensure that patient care is not compromised and that the environment and equipment are correctly cleaned without undue delay. At present, cleaned rooms and bed spaces are routinely inspected by eye before admission in United Kingdom and most state-run health care systems. Less subjective methods of cleanliness assessment have yet to become widely incorporated into routine monitoring of the health care environment other than for research purposes (129).

Microfiber versus Cotton

Most hospitals prefer cotton-based cloths and mop heads for continued use, since these can be repeatedly washed at high temperatures (>90°C). There are many types of cleaning cloths, however, with microfiber products now proving popular among cleaning staff (142). Ultramicrofiber (UMF) cloths are made of a combination of polyamide and polyester, which absorb particles of soil through static attraction. Dust and organisms become firmly attached to the synthetic fibers and tend to persist within the cloth throughout the cleaning process. A range of different types of damp microfiber cloths were recently evaluated for their ability to remove pathogens, including *C. difficile* spores, MRSA, and *E. coli* (143). Single-use damp microfiber cloths demonstrated a mean log₁₀ reduction of 2.21 after cleaning, with smaller reductions obtained after repeated use on a series of contaminated surfaces.

An *in vitro* study was performed to evaluate and compare reusable (rayon fiber) J-cloths against UMF cloths for eliminating Acinetobacter spp., MRSA, K. oxytoca, and C. difficile spores from hospital surfaces (144). UMF cloths were significantly better than J-cloths for removing pathogens from tiles, new and used laminated worktops, and stainless steel surfaces. These cloths generally eradicated most, if not all, cultivable bacteria or *C. difficile* spores from the surfaces tested, while standard J-cloths did not. This included used laminate surfaces, which can provide hidden reservoirs for bacteria within surface microfissures (144). The results differed from those of an earlier study using ordinary microfiber, which reported less striking cleaning abilities depending upon type of product tested (142). The authors attributed the divergent results to dissimilar structures and lengths of fibers in the cloths used in the two studies, but there were differences in study design (144). The previous study used microbiological methods and ATP bioluminescence to assess cleaning efficacy, whereas the later

UMF study tested surfaces using ATP bioluminescence only (142, 144).

In contrast, another study has showed that microfiber cloths are only marginally more efficient for removal of soil and associated microbes than cotton cloths in the presence of organic matter (145). For surfaces without soil, no significant difference has been found between cotton and microfiber cloths (142, 143, 145). Thus, the final choice between traditional and microfiber cloths for cleaning purposes rests with those with purchasing responsibilities. It is hoped that these individuals take advice from staff who actually perform the cleaning themselves. Microfiber products are too expensive for single use, so continued use should be subjected to a cost-benefit analysis. Furthermore, decontamination of the cloths is required after cleaning, since pathogens, including spores, may adhere to the synthetic fibers. Exposure to bleach and other disinfectants potentially damages some microfiber products and shortens their life span, so it is important to check manufacturers' recommendations before purchase (146).

Contamination of Cleaning Equipment and Liquids

Comprehensive cleaning schedules are seriously compromised if cleaning equipment or liquids are contaminated. Poor choice of cleaning methods or products or inadequate maintenance of equipment will result in environmental contamination of the very surfaces that need attention. There are numerous examples of cleaning cloths, including those made of microfiber, that merely distribute organisms across surfaces instead of removing them (26, 120, 142, 147–149). Enterococci, including VRE, seem to be particularly difficult to eliminate from contaminated cloths (51, 147, 148).

Cleaning equipment is also vulnerable to contamination from hospital pathogens and this encourages further dispersal throughout the hospital environment (24, 82, 150, 151). Disinfectants are supposedly better at killing environmental organisms than detergent-based agents, but some pathogens are able to survive exposure to specific biocides (152). Both multidrug-resistant *S. marcescens* and extremely drug-resistant strains of *K. pneumoniae* have demonstrated increasing tolerance to chlorhexidine (153, 154). Other cleaning fluids can become contaminated with Gram-negative bacilli during use, with some formulations apparently encouraging acquisition of resistance elements by Gram-negative organisms (126, 155).

Microorganisms will exploit an inadequately cleaned niche to exchange genetic material coding for antimicrobial resistance and other survival mechanisms (156–158). This could include resistance or tolerance to disinfectants. Once established, these hardy strains may ultimately infect debilitated patients (157). Hospital wastewater has been shown to harbor KPC-2-producing *K. pneumoniae*, suggesting widespread contamination throughout the health care environment (159).

Benefits of Physically Removing Soil

While most hospitals would use disinfectants for cleaning rooms or areas around colonized or infected patients, there have been a number of recent papers that suggest that physical removal of bioburden, rather than biocidal activity, is integral to the cleaning process (160–164). This was first suggested over 50 years ago, when it was shown that reduced surface contamination following disinfectant exposure appeared to be due to the cleaning activity, rather than the killing activity, of the products tested (165, 166).

How should we clean clinical equipment?



All three protocols decreased MRSA surface load by >99% from 10-14 cfu/cm² to 0.1 cfu/cm² (p<0.001)

FIG 2 How should we clean clinical equipment? This figure shows data from a study examining three different methods for cleaning a dental chair. Cleaning (wipe-rinse method) using a sodium lauryl sulfate-based detergent demonstrated equivalence to use of a disposable barrier and bleach disinfection for reducing MRSA contamination on a dental chair (164). (Photo courtesy of S. Petti.)

Two studies published in 2012 found that norovirus (human and MNV1 strains) could be substantially reduced on hard surfaces after wiping from one to six times using a range of inocula and material wipes (121, 149). This supports the increasingly popular premise that physical removal could challenge routine use of disinfectants for controlling surface microbes (163, 164) (Fig. 2). This may be related to the fact that the presence of organic soil on a surface will impede the microbicidal activity of a disinfectant, but this is not the only explanation. Frequent physical removal of bioburden using detergent-based cleaning methods needs to be compared and contrasted with application of biocides for cost benefits as well as longer-term efficacy and environmental issues (167).

There is no doubt that detergents are less toxic than powerful disinfectants as well as less likely to encourage accumulation and dispersal of tolerant or resistant genes among hospital strains (167, 168). A study performed recently demonstrates the effect of detergent-based cleaning over a 48-h period for high-risk hospital surfaces (133). The study measured the total bioburden and presence of *S. aureus*, including MRSA, using standardized methods. The results suggest that wiping over near-patient surfaces once a day with single-use detergent wipes might be sufficient to protect patients from environmental pathogen reservoirs in a nonoutbreak situation. Disinfectant wipes add cost without necessarily greater efficacy at pathogen removal (169).

AUTOMATED DECONTAMINATION DEVICES

Given increasing awareness of the role of cleaning, recent innovations have tried to improve the scope and quality of cleaning practices in the health care environment (170). There is a continuing risk of transmission from pathogens within residual bioburden if surfaces remain uncleaned or receive inadequate cleaning. Carling and coworkers applied a transparent gel to selected surfaces in more than 1,000 patient rooms in 23 acute-care hospitals before cleaning in order to assess the quality of housekeeping services. The gel is easily cleaned, difficult to detect, stable, and nontoxic, and it fluoresces when exposed to hand-held UV light (170). If UV

inspection detects persistent gel on a surface after cleaning, it is assumed that the site did not receive sufficient cleaning attention. The overall cleaning compliance following gel application was only 49% (range, 35% to 81%), expressed as a percentage of evaluated surfaces. With this sort of information, it is not surprising that several manufacturers are developing automated room disinfection units that demonstrate superior decontamination of environmental objects and surfaces. These systems deliver various microbicidal products, including germicidal light, hydrogen peroxide, steam, and ozone (171–175).

Automated technologies may offer enhanced decontamination, but they cannot replace routine daily cleaning. Organic soil, liquids, waste, and litter must still be removed from floors and surfaces before disinfectant agents are released. Furthermore, these machines can usually be used only for terminal or discharge cleaning because the products are either too toxic for patients (e.g., hydrogen peroxide), constitute a safety risk (e.g., steam), or are better suited to work in empty rooms (e.g., UV light).

Steam Cleaning

Steam vapor machines are rapidly effective against a wide range of pathogens, notably VRE, MRSA, and Gram-negative bacilli, including *P. aeruginosa*. Initial inocula of 7 log₁₀ selected organisms are reduced to undetectable levels in less than 5 s following exposure to steam (176). The total surface bioburden from hospital surfaces is decreased by more than 90%, along with almost complete elimination of pathogens (177). While solid rubbish should always be removed before this type of disinfection, steam can be directly applied onto a wide variety of soft and hard surfaces without prior cleaning (175).

Reports of the efficacy of steam cleaning are few in the literature, but there are examples of benefit from using steam in both routine and outbreak situations. An outbreak of norovirus occurred on two wards at the same time in an Australian hospital (178). Two different cleaning protocols were instituted for each ward: one ward received detergent and bleach (1,000 ppm sodium hypochlorite plus contact time of 10 min) as a sequential 2-step method, and the other was provided with steam technology. The steam component was applied using microfiber cloths and mops for terminal cleaning. The advantages from using steam were fewer cleaning hours, no toxic chemicals or dry-cleaning costs, and 90% less water consumption. The end result was visually superior, with clear support from cleaning staff. Microfiber-steam technology also proved to be a highly effective method of decontamination in an outbreak situation, with the same advantages as reported for routine cleaning (161, 178).

Concern has been expressed over some aspects of the steam technology for routine hospital cleaning (179). The use of steam to decontaminate hand touch sites such as knobs, buttons, switches, and computers, including those on electrical appliances, presents obvious practical problems. If a hospital implements steam-based cleaning in preference to other methods, there is a risk that these high-risk sites might miss out on appropriate cleaning. It is also the case that the temperature of steam at delivery may rapidly dissipate depending upon the type and conductivity of exposed surfaces. This has implications for the length of time that surface organisms are exposed to applied steam. A previous study showed that steam cleaning of curtains on a disused ward proved difficult to implement because there was no indication which areas had

received sufficient steam exposure, and pathogens were recovered before and after the process (125).

Ultimately, steam delivered to surfaces turns into water. Residual moisture constitutes a risk of slips and falls for patients, staff, and visitors, although superheated steam is less likely to leave water on exposed floors and other surfaces (179). Steam cleaning presents further problems when cleaning a crowded ward, because there may be difficulties gaining access to sites beside a bed-ridden patient. There are also time pressures for busy wards, which compromise effective cleaning of a bed space if cleaners have only minutes to deliver the service. In addition, carelessly handled equipment represents a continued risk of burns and scalds for both handlers and persons nearby, including patients. Inhalation of the vapor could potentially aggravate breathing problems in staff or patients with respiratory conditions (179).

Some hospitals have adopted a rolling program of steam cleaning commodes, beds (nonelectrical), and other furniture in nonclinical areas. Steam also offers a useful cleaning strategy for public toilets in hospitals and elsewhere. Steam systems should generally be used only in well-ventilated areas, since repeated buildup of condensate could influence the environmental bioburden as well as damage the internal fabric. Depending upon the type of equipment and surfaces selected, there remains a need for comprehensive risk assessment of aerosolized pathogens from the vaporizing process (179).

Ozone

Ozone is a potent oxidizing agent which has limited impact on bacterial spores and fungi but is highly effective against vegetative bacterial cells (180, 181). While it is relatively cheap to produce, it is both toxic and potentially corrosive for metals and rubber despite rapid dissociation into oxygen. There are consequently only a few studies reporting its use in health care settings (171).

One recent study demonstrated benefit when ozone was incorporated into laundry decontamination. A hospital laundry system using ozone resulted in a 5 log₁₀ reduction of E. coli and total coliform count present in rinse water (182). Two other studies have reported that ozone has potential as a gaseous decontaminant for controlling environmental C. difficile, with various results. The first showed that C. difficile could be reduced by >4 log₁₀ on various surfaces using a standard delivery of 25 ppm ozone for 20 min at 90% relative humidity (183). The second found that a 3 log₁₀ reduction in C. difficile spores was obtained following 25 ppm ozone for 75 min (184). In a domestic setting, an estimated concentration of 12 ppm was needed to eradicate MRSA from home surfaces (180). An earlier study used a gaseous ozone generator for decontaminating hospital side rooms previously occupied by MRSA patients (174). Concentrations of 0.14 ppm were achieved for different lengths of time, which failed to eradicate environmental MRSA and also initiated respiratory symptoms among exposed staff.

UV Light

UV irradiation has been investigated as a potential decontaminant against environmental pathogens, including disinfection of surfaces, instruments, and air (185). UV light severs the molecular bonds in DNA at specific wavelengths in order to exert its microbicidal effect. UV-C light has a specific wavelength found between 200 and 270 nm (usually 254 nm), which itself falls within the germicidal segment of the electromagnetic spectrum (200 to 320

nm). Investigations of the effects of UV irradiation should consider the interaction between several different parameters, notably, time of exposure, lamp position in relation to the irradiated surface, barriers between the light source and target surface, intensity of emitted light, and extent and flow of air movement. These could all influence the overall effect of UV-C irradiation on surfaces.

There have been several studies in the last few years examining the effect of UV light as a potential decontamination strategy for health care environments. Nerandzic et al. described the effects of a fully automated UV-C system against hospital pathogens (186). The device was tested in the laboratory and patient rooms and was shown to significantly reduce C. difficile, VRE, and MRSA contamination on frequently handled hospital surfaces. The same group investigated a hand-held version delivering UV-C irradiation (185 to 230 nm) against pathogens in the laboratory, in patient rooms, and on surfaces of items such as keyboards and portable medical equipment located outside patient rooms (187). While the device significantly reduced C. difficile and MRSA, organic matter on hospital surfaces that were not manually cleaned before irradiation clearly impeded the overall effect. This means that routine cleaning practices should still be carried out, even if a hospital chooses to implement routine decontamination using UV technology.

Another study describes the decontamination effect of a portable pulsed UV light device and its impact on work load when introduced into a hospital ward (188). Using pulsed UV for routine once-daily disinfection of ward surfaces halved the number of housekeeping hours compared with the time taken for manual disinfection using alcohol wipes. Other studies have shown that UV-C systems can reduce vegetative bacteria by >3 to $4\log_{10}$ within 20 min after inoculation onto a carrier, although 35 to 100 min of irradiation is required to reduce *C. difficile* by >1.7 to $4\log_{10}$ (189, 190). When surfaces were not directly in line with the UV light source, the systems were not quite as effective.

Some authors have stated that although UV light is microbicidal, it should not be used as a first-line intervention for decontamination but should be considered for use as a supplementary strategy depending upon specific needs, e.g., high or escalating HAI rates (185). There are several factors to consider before implementing routine UV-C technology; these include overall costs, installation, hospital layout and design, integration into housekeeping services, management of UV operation (including bulb choice and longevity), and traditional cleaning and disinfection practices. UV light is significantly less effective for sites around corners or shielded by solid items that challenge penetration by light rays. It may also damage plastics and polymers used in the health care environment if repeatedly exposed. At this time, more work is required to evaluate the costs versus benefits, safety, and incremental advantages of UV devices for controlling health careassociated infections.

HINS

High-intensity narrow-spectrum (HINS) light is another light-based disinfection method that has shown wide-ranging microbicidal activity (191). HINS light utilizes a narrow bandwidth of high-intensity visible violet light with peak output at 405 nm. The microbicidal mechanism is different from that of UV-C, in that microbial inactivation is thought to be due to photoexcitation of porphyrin molecules within bacterial cells. This encourages the

production of singlet oxygen as well as other highly reactive bactericidal compounds (192). One study has evaluated the overall effect of HINS light for decontaminating the clinical environment, but further work is needed to investigate any benefits on HAI rates from this technology (172).

Hydrogen Peroxide

Several systems which produce hydrogen peroxide (HP) in different formulations (e.g., HP vapors and dry aerosols) have been studied for their potential to decontaminate environmental objects and surfaces in hospital rooms. HP systems are effective against *M. tuberculosis*, MRSA, viruses, sporeformers, VRE, and multiresistant Gram-negative bacilli, including *Acinetobacter* spp. (53, 73, 193–195). Using a before-and-after design, Boyce and coworkers showed that introducing HP systems onto high-incidence wards was associated with a significant decrease in rates of CDI (196).

HP systems appear to offer reliable microbicidal activity against most, if not all, hospital pathogens, but a number of problems have been raised in association with these systems. Risks of accidental exposure of people, animals, and plants continue, with repeated use of HP liable to encourage erosion of some plastic and polymer surfaces or items used in health care environments (197). Disinfection is impeded by residual debris, such as organic soil, liquids, and waste, as well as surface properties, such as linen and other soft materials. The equipment needs to be carefully positioned in order to facilitate optimal exposure, but this may compromise overstretched clinical staff. Without additional support, there may not be sufficient time to coordinate such preparation or perform it adequately.

Like UV-C systems, HP devices are universally expensive, cannot be used in occupied rooms, and require trained operators. They also need planned integration into decontamination and housekeeping schedules. Effective HP disinfection may take several hours to complete a full cycle, which contrasts with the time taken for traditional discharge cleaning (197). Delivering HP decontamination may prove difficult in a hospital running at 100% bed occupancy, since any restrictions in bed turnover time could easily have an impact on admission capacity (197, 198). Rooms cannot be easily closed in today's crowded hospitals, let alone multibed bays, complete wards, or specialist units offering 24-h emergency care.

Pottage et al. compared MRSA resistance to HP against commercially available spore indicators inoculated onto stainless steel coupons (199). The recovery of MRSA from test coupons was between 1.5 and 3.5 \log_{10} higher than the quantity of *Geobacillus stearothermophilus* spores recovered after exposure (P < 0.05). The greater resilience displayed by MRSA may have been due to production of catalase, which is presumed to break down HP, leading to reduced efficacy. This highlights the fact that sterilization competencies achieved using standard biological indicators cannot always be extrapolated to other organisms. Preliminary cleaning of surfaces should always be performed to remove the original bioburden, just as specified for UV light (171, 187).

Comparison between UV Light and Hydrogen Peroxide Systems

HP and UV systems have inevitably drawn comparisons. UV-C devices cannot eliminate bioburden on surfaces that are not directly in line with emitted light rays, but they do offer a faster

decontamination cycle. This reduces the time period that the room is unavailable for patient admission (200). HP and UV devices decrease microbial contamination in patient rooms, with HP vapor delivery apparently significantly better at removing bacterial spores. These differences may be influenced by exposure time and/or intensity of emissions for both systems and require further clarification. Whether superior sporicidal activity is clinically important is unclear, since environmental screening has shown that the quantity of spores is relatively low on surfaces near patients with C. difficile infection. This is also true for VRE and MRSA. There are two recent studies, however, that both report a reduction in C. difficile incidence among patients, the first after introducing a pulsed UV system into a community hospital and the second after using pulsed UV in a large academic medical center (201, 202). The latter paper also reported an overall decrease in the number of patients acquiring multidrug-resistant organisms despite missing a quarter of opportunities to apply the device after patient discharge (202).

Innovative technologies offer an alternative strategy for environmental hygiene purposes, but their logistical complexities, aside from costs of equipment, training, management, and personnel, make it imperative that objective, controlled, and independent studies be performed in order to establish overall costs versus benefits (203). Furthermore, studies have hitherto concentrated on efficiency of surface disinfection without specifically examining the effects on airborne pathogens. Rapid disinfection cycles may well sterilize hard surfaces without eliminating viable organisms surviving in the air (41). Concern has been expressed by several authors over the premature incorporation of these systems into routine decontamination schedules (171, 173, 197, 203, 204). Cost-effectiveness studies would help health care managers choose the most appropriate system for their facilities based on evidence rather than advertising (197).

ANTIMICROBIAL SURFACES

While regular and conscientious cleaning is a necessity for eliminating pathogens, it is not the only mechanism for keeping surfaces free from microbes. There are some high-tech solutions currently receiving attention, including the so-called "self-sanitizing" surfaces. The technology was first suggested in 1964, but given the long-held view that hospital surfaces were not relevant for HAI control, the potential use of antimicrobial surfaces has only just begun to generate discussion (205, 206). It is possible that treating or coating hospital surfaces liable to contamination by pathogens could kill or inhibit microbes in order to disrupt transmission to patients. Hard metals such as copper and silver have long been investigated for their antimicrobial properties, and now novel technologies such as light-activated titanium dioxide-containing surfaces are attracting attention (207–209).

The development of effective antimicrobial surface coatings could impinge on the risk of cyclical transmission of pathogens between surfaces, hands, and air (206). These coatings might deter the accumulation of microbial bioburden on a surface without additional or increased frequency of cleaning and would therefore contribute toward hygiene practices in the clinical environment. Stopping a surface from functioning as a microbial reservoir effectively reduces the risk of onward transmission in health care environments. The risk from person-to-person transmission remains, but this may be tackled by barrier nursing and hand hygiene programs for staff, visitors, and patients. Self-sanitizing sur-

faces have the ability to supplement manual cleaning, which is itself dependent upon operator time, choice, and ability and thus subject to considerable variation (170, 210).

There are several types of antimicrobial surfaces. A comprehensive review of these surfaces has been written by Kristopher Page and colleagues, from which the following classifications have been extracted (206). Antimicrobial surfaces can be placed in two main categories: first, antiadhesive coatings, and second, antimicrobial coatings and surface technologies. The latter category contains examples such as bacteriophage-modified surfaces, polycationic surfaces, and light-activated coatings (206).

Antiadhesive Surfaces

One approach toward inhibiting microbial contamination is to engineer a surface that prevents microbial adhesion to the device or surface. This can be achieved by applying a layer of polyethylene glycol (PEG) directly onto the surface (211). PEG-coated surfaces create a hydrophilic interaction against hydrophobic bacterial cells, which impedes microbial attachment. The dynamic properties of surface-bound PEG chains also make it more difficult for microbes to become attached. Diamond-like carbon (DLC) films similarly repel microbial adhesion and have been used as nontoxic surface coatings for devices such as joint prostheses or stents (212). Easy-clean surfaces are either exceptionally hydrophilic or hydrophobic, with strongly hydrophobic coatings repelling bacteria to a much greater extent than glass controls or other commercial coated glass products (213). Hydrophilic surfaces encourage water sheeting and ease of cleaning. Polymers can be manufactured with zwitterionic head groups, which are also useful for inhibiting bacterial adhesion and biofilm formation (214). The zwitterionic head attracts a large amount of water and makes the material hydrophilic. All these surfaces and easy-clean technologies are compromised in one respect, however, which is their lack of inherent biocidal properties.

Antimicrobial Coatings

Triclosan. There is a wide range of antimicrobial coatings, some of which are commercially available while others exist only at the research stage. Currently available products either are based on organic antimicrobials impregnated into a specific product, e.g., Microban (triclosan), or rely on inorganic antimicrobials such as ionized silver (Ag⁺) or copper in different formulations (215). Surfaces that utilize diffusible antimicrobials could potentially induce microbial tolerance or even resistance, because the products continually leach out active compounds into the environment (206). With Microban products, the antimicrobial diffuses over the surface to exert antimicrobial activity, making it nonpermanent. About 75% of antibacterial liquid soaps and 30% of bars also use triclosan, which was used only in hospital settings until the 1990s. Widespread use may be linked with its presence in nasal secretions of healthy people, where it appears to be associated with S. aureus nasal colonization (216).

Much concern has been expressed over the development of resistance to triclosan (217). It has been inferred that triclosan encourages the production of poisonous dioxins following exposure to UV light (218). Recently, the FDA (U.S. Food and Drug Administration) announced a new position on antibacterial soap, including those products containing triclosan (219). Manufacturers must show both that their products are safe and that their use is superior to simple washing with conventional soap and water,

or they will have to remove them from sale before 2016. This should also apply to impregnated surfaces. It is possible that the costs of antibacterial products outweigh any potential benefits, unless proved otherwise.

Silver. Both the Greeks and the Romans favored drinking vessels made of silver to make water potable. It is thought that silver ions (Ag⁺) bind to thiol (–SH) groups present in microbial enzymes and proteins, inactivation of which produces the desired antimicrobial effect (208). However, Ag⁺ coatings do not maintain permanent activity despite initial effectiveness. Bacteria can become tolerant or even resistant to silver coatings or products (220). They therefore rely on additional diffusible antimicrobials, such as rifampin, to which microbes may also become resistant. Silver has been incorporated into various products, including coatings and textiles (221–223). Some coatings demonstrate good antibacterial activity against planktonic *Staphylococcus epidermidis* and *A. baumannii* and have been used to inhibit environmental contamination as well as colonization of implanted medical catheters and other devices (223).

Copper. Copper is also toxic to microbes, and there have been several studies examining the antimicrobial effect of coating hospital surfaces with copper (207, 224). There is no doubt that both copper and copper alloy surfaces demonstrate a profound antimicrobial effect. There is even one study that attributes a reduced rate of hospital-acquired infection to the installation of copper coatings onto near-patient surfaces (45). The study itself reported only some of the overall data, which detracted from the overall conclusions, but it is clear that further research in this area is warranted (225).

Bacteriophage-modified surfaces. There have been recent attempts to apply bacteriophages to surfaces in order to control bioburden (226). Antibiotic resistance capabilities do not necessarily protect bacteria from attack by phages. Since only one phage is required to infect a host cell in order to initiate multiple phage production, this approach could represent an efficient way of disinfecting a surface. There are a number of complications, however, mainly due to the inherent specificity of a phage for a particular species of bacteria (206). While this forms the basis of targeted in vivo therapy, it is less useful for open surfaces due to the wide variety of dynamic bioburden, not necessarily bacterial. A mixture of phages should be applied in order to increase the spectrum of activity, and this would exclude nonbacterial organisms as well as rare or unusual pathogens. Furthermore, large or uneven surface areas may impede the distribution of phage solutions and deter them from reaching their appropriate bacterial target (226).

Phage stability in the environment and storage conditions also represent important issues for study, as well as the potential for phage resistance. Phage formulations and treated surfaces need to be continually monitored and revised in order to remain effective, which will complicate regulatory approval (206). Finally, the phage concentration of a solution required for effective decontamination should be carefully considered, along with the most appropriate incubation time for a specific phage and target (226).

Polycationic antimicrobial surfaces. Surfaces treated with hydrophobic negatively charged polycations kill bacteria by causing physical damage to the cellular envelope. Hydrophobic polymer chain coatings attract bacteria toward the treated surface, resulting in puncture of the cell wall and subsequent cell death. Recent examples of this type of surface coating include the polyethyleneimines (PEIs) (227). While PEI coatings are thought to be per-

manently microbicidal, their longevity, biotolerance, and mechanical stability have not been widely investigated. There is no information as to whether these coatings will be able to withstand routine wear and tear in health care settings, including cleaning practices and disinfectant exposure (206).

Light-activated antimicrobial surfaces. Another surface decontamination strategy is to use a coating that produces reactive radicals. Biotoxic radicals, unlike antimicrobial agents, do not target a specific microorganism but exert nonselective effects toward a range of microbes (228). This means that they avoid the potential problem of an organism developing resistance to a specific treatment. There are two main types of coating that produce reactive species and consequently display antimicrobial properties. The first is based on a photosensitizer immobilized within a coating, and the second is a coating containing a titanium dioxide (TiO₂)-based catalyst (209, 229, 230). Both of these are classified as light-activated antimicrobial agents.

Various modes of action have been investigated, specifically the mechanism of photocatalysis and how this results in microbial killing (206). The effectiveness of TiO₂ as a photocatalyst is based on the rate of production of hydroxyl radicals at the surface of the semiconductor, although the energy of the light illuminating the surface is also important. There have been some attempts to examine the performance of these coatings in health care environments. In one study, TiO2 efficacy in preventing MRSA contamination in a clinical environment containing MRSA patients was only 17.8% (231). The study did show, however, that environmental contamination was higher for untreated surfaces (12.1%, versus 4.4% for treated surfaces) and also higher for ad hoc samples taken from an environment exposed to MRSA as opposed to a non-MRSA environment (14.1% versus 3.5%, respectively). Disinfection of a surface by photocatalyzed reactions may be an alternative and less toxic approach to using chemical disinfectants, but it is important to be certain that these coatings demonstrate long-term efficacy in working health care environments.

Current Concerns over Antimicrobial Surfaces

The utility of antimicrobial surfaces needs careful consideration before widespread adoption (17, 232). Current evidence has shown that these surfaces produce a moderate microbicidal effect only (<2 log₁₀ pathogen reduction), with no studies yet investigating efficacy against pathogens such as C. difficile spores and norovirus. The resources required for installation in health care settings and overall cost-effectiveness are unknown. There is insufficient information on durability and whether antimicrobial activity is affected by humidity, temperature, cleaning frequency, and/or the presence of an organic load (233). There are ongoing concerns over possible toxicity, resistance, and allergenic properties (17, 218, 234). Finally, the relative contribution of self-disinfecting surfaces toward hand contamination and consequential risk of cross-transmission has not been established (232). We do not know which sites, surfaces, and clinical equipment in patient areas should be, or could be, coated with an antimicrobial product. It is true to say, however, that continued research on these surfaces is needed and will no doubt attract much interest from business and industry in the future. There has already been a call for scientific standards for antimicrobial surfaces in view of the rapidly expanding technologies and potential importance of these products (235).

HOW TO MEASURE CLEANLINESS

There are a number of scientific methods in use for measuring environmental soil, since visual inspection cannot accurately determine the infection risk for patients (129, 236, 237). The definition of "clean" requires a validated and risk-assessed strategy to establish a state of "cleanliness," rather than the subjective assessment currently provided by visual inspection and clipboards (129). Microbiological and chemical (ATP bioluminescence) techniques have long been incorporated into a comprehensive assessment framework utilized by the food industry, and these techniques are now being tested in hospitals (17, 42, 236–239). Measurements from these methods have furnished a range of tangible values that can be modeled against the infection risk for patients over time. Collecting data using microbiological and chemical tools provides an opportunity to choose an appropriate benchmark for routine surface monitoring. This benchmark should signify whether hospital cleanliness levels indicate a clinical infection risk or not (42, 240). Health care staff, including housekeepers, would welcome an evidence-based cleanliness standard, thus allowing them to review, change, or target cleaning practices before an outbreak becomes inevitable (1). Managers would benefit from established benchmarks, since they would be able to audit, monitor, and defend practices in both routine and outbreak situations.

Microbiological Methods

Current microbiological standards include an overall aerobic colony count and specific pathogen count for defined surface areas health care environments (129). Aerobic colony counts of <2.5 to 5 CFU per cm² on hand touch sites and <1 CFU/cm² hospital pathogen (e.g., MRSA, VRE, *C. difficile*, etc.) have been proposed and tested as microbiological benchmarks (42, 43, 236–238, 241). The two benchmarks appear to be related, in that higher levels of aerobic colonies on hand touch sites are more likely to be associated with the presence of *S. aureus* and MRSA (237). The standards have been used to systematically measure soil in several hospital studies but have not yet been validated for routine monitoring (42, 133, 242, 243).

Similar counts for food preparation surfaces form the basis of the monitoring framework set up by the food industry (129, 132). Retail and food manufacturers, plus a variety of other agencies, use microbiological standards based on the presence or absence of indicator organisms, identification of which alerts the agency to a potential health risk from the medium monitored (132, 244, 245). These standards also incorporate overall counts of nonpathogenic flora, because the organisms of interest are widely spread throughout time and space (129). The most reliable indicator of environmental hygiene in health care premises is the presence of coagulase-positive staphylococci, because ubiquitous human carriage and frequent human traffic encourage risk of contamination. Studies investigating the application of microbiological standards in health care environments have selected both *S. aureus* and MRSA to help monitor cleanliness (1, 2, 42, 43, 241, 246).

ATP Bioluminescence Systems

ATP bioluminescence systems are provided with various benchmarks depending upon make and model of luminometer and the environment to be monitored. The benchmark levels range from 25 to 500 relative light units (RLU) for 10- to 100-cm² health care surfaces (238, 241, 247). Studies have suggested that some systems

are not sufficiently sensitive to detect very low microbial counts (<10 CFU/cm²), which is of concern given the low numbers of pathogens required to initiate infection (Table 1) (248, 249). Other studies have investigated possible associations between ATP and microbiological data by systematically measuring both data sets from the same surfaces. One study found that benchmark categories of 100 RLU and microbial growth of <2.5 CFU/cm² were only loosely related, since approximately 60% of combined data sets agreed as to whether a surface should pass or fail (241). Another examined colony counts and ATP values independently against cleaning performance using fluorescent markers (250). The data presented suggest that ATP monitoring is more useful for detecting the need for cleaning attention, whereas microbiological screening provides an indication of the quality of cleaning (250). It is clear that more studies are required in order to establish the best method for monitoring hospital surfaces in the routine situation (249, 251).

ATP measurements can be hugely inflated by disinfectants, microfiber products, food and drink spillages, and synthetic plastics used in cleaning and laundry services (236, 251, 252). Chosen benchmarks should reflect the risk of infection for different types of patients accommodated in different clinical areas. Sites and surfaces in outpatient clinics, hospital corridors, and storage areas do not necessarily provide the same level of infection risk as surfaces in a bone marrow transplant unit or hand touch sites beside an ICU patient. After these benchmarks have been established, routine monitoring should be able to highlight problem areas or trends illustrating the dynamic balance between hospital cleanliness, staff deficit, and workload. Most importantly, awareness of a sudden accumulation of soil might initiate extra cleaning before patients are exposed to a risk of infection or even an outbreak (42, 238, 240). As previously stated, several studies have already shown the association between bioburden on health care surfaces and HAI rates, whether due to overall HAI or specific pathogens (42, 43, 45, 63, 74, 253).

HOW TO MEASURE CLEANING

Fluorescent Markers

There are alternative ways of assessing the health care environment, notably monitoring the efforts of cleaning staff rather than measuring residual bioburden on surfaces. Most environmental failures are likely due to personnel themselves, not products or practices (254). Assessment of the cleaning process can be introduced by using educational strategies, direct and indirect cleaning inspections, observation, scientific monitoring, and feedback to staff (17, 58, 170, 247, 255). Any form of environmental monitoring is quickly noticed by housekeeping staff, although the effect can wear off without continued feedback or education (17). Inoculation of key sites using invisible fluorescent markers for later inspection virtually always improves overall cleaning compliance, with a reduced prevalence of hospital pathogens (255-257). However, cleaners become aware of this type of monitoring, search out fluorescent marks, and then target these for cleaning to the detriment of other sites and surfaces (258). Although more research is needed, a few studies have indicated that the use of fluorescent markers is linked with decreased transmission of hospital pathogens (51, 259).

ATP Bioluminescence Systems

Tangible values and trends over time from bioluminescencebased ATP data have the advantage of immediate and potentially longer-term feedback for housekeeping staff (260). The use of ATP monitoring appears to have a pronounced effect on cleaners, especially when they receive educational programs at the same time (247). Similar to the case for fluorescent marking, housekeeping staff react quickly to an environmental monitoring program because they are concerned that their jobs may be at risk (17, 58).

Observation, Supervision, and Education of Housekeeping Staff

Several other studies have demonstrated different results after instituting direct observation, supervision, and education of staff as they clean, again often showing reductions of important hospital pathogens (257, 261–264). There is a concern that these interventions might lose impact over time, since cleaning is physically demanding, poorly paid, and subject to inadequate staffing (17, 265). Furthermore, there tends to be rapid turnover among janitorial and housekeeping staff, and this may be related to higher sickness levels as well as dissatisfaction with pay, status, and conditions (17).

Ongoing training, education, and continual evidence-based reassessment are required as an important part of staff management. It is hoped that the overall status of housekeeping staff improves in parallel with the recognition of the importance of basic cleaning in health care environments. Specialized cleaning activities can be agreed to and implemented for staff who wish to assume greater responsibility and are prepared to undergo relevant training and assessment. Selected housekeeping staff could potentially manage the decontamination of clinical equipment, traditionally the remit of clinical staff, thus releasing more time for the latter to care for patients. Perhaps the creation of a new training framework for different levels and competencies of cleaning staff would help raise the status of cleaners, as well as focus attention on the cleaning resources required to keep health care environments safe for patients.

DISCUSSION

There is no easy way to clean a hospital or to keep it clean, however we define "clean." Removing visual and invisible dirt from the hospitals of today and for the future requires sufficient trained staff, ongoing monitoring, measurement of bioburden, education, constant upgrading of practice, and two-way communication between those responsible for cleaning and those responsible for infection control. The risks of cross-transmission are exaggerated by heavy workload, understaffing, high bed occupancy rates, and rapid bed turnover (266). Poor ventilation, clutter, and inappropriate storage further compound the ability to clean surfaces properly and keep them clean (24). Furthermore, in an era of cost cutting, those with cleaning responsibilities cannot hope to decontaminate all high-risk surfaces as often as required when a hospital is full to capacity and patients with attendant microorganisms are transferred between wards (and hospitals) day and night (17, 267).

Current Unanswered Questions

While most would agree that keeping hospitals "clean" and prioritizing surface cleaning around the patient are of paramount im-

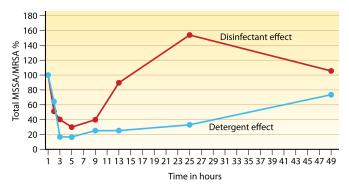


FIG 3 Effect of detergent and disinfectant cleaning on total Staphylococcus aureus (methicillin-susceptible S. aureus [MSSA] and methicillin-resistant S. aureus [MRSA]) recovered from hand touch sites on a 30-bed ward over 48 h. This figure shows the effects of detergent (blue line) and disinfectant (red line) on surface S. aureus and MRSA from baseline levels over 48 h on a 30-bed acute ward. Both types of cleaning rapidly reduced the overall staphylococcal burden, but recontamination occurred more rapidly after disinfectant exposure. The sites monitored were bedside locker, bed frame, and overbed table, and each 48-h period for each type of cleaning was repeated three times (276). (Adapted from reference 268.)

portance, there are several key questions to which we do not yet know the correct answers. These can be listed as follows. (i) How important is the choice of cleaning fluid, whether detergent and/or disinfectant? (ii) How much better is microfiber than traditional cloths? (iii) When should we use bleach, and when should we not? (iv) When should we consider the use of automated systems? (v) How should we monitor cleaning? (vi) How should we monitor cleanliness? (vii) Are current specifications targeting the most contaminated sites? (viii) How often should we clean an occupied room or bed space?

The debate over detergent- or disinfectant-based cleaning in the routine situation continues to rage unabated. Ignorance about the effects, short and long term, of cleaning agents persuades managers to choose powerful kill-all fluids or gases for their hospital as protection against pathogens and lawsuits. Microbiologists and environmentalists argue that the removal of dirt should be achieved without resorting to expensive toxic agents, which may themselves encourage the appearance and persistence of resistant pathogens in habitually exposed environments. Regarding the proliferation of automated dispersal systems for decontamination of surfaces, there may be unintended consequences of such new technologies, quite apart from the expense involved in introducing them. Advertising and marketing are much less costly than

The fact that physical removal may be just as good at removing soil as disinfectants is supported by several recent studies and emphasizes the need for more work in order to avoid environmental and human toxicity from potent disinfectants (160–164) (Fig. 2). A recent study suggests that the effect of detergent cleaning on surface S. aureus and MRSA lasts longer than the effect seen after disinfectant exposure (268) (Fig. 3). Aside from this, first-line use of detergents for routine cleaning saves money as well as negating any risks from tolerance or resistance among pathogens due to disinfectants (197). Hospitals in the United Kingdom routinely use detergent-based cleaning for general surfaces and do not seem to experience the same levels of MDR Acinetobacter and VRE as reported by disinfectant-using hospitals in other countries (3, 19, 73, 80, 269). It is true to say that cleaning is not the same as disinfection, although the two terms are habitually interchanged (270).

There appears to be a link between HAI rates and environmental bioburden, although as yet only a few studies have reported this and even fewer have investigated it (42, 43, 45, 63, 74, 253). More work on this relationship is urgently required, since a measurable association offers tangible proof for the role of the environment in HAI risk. It also justifies the setting of scientific standards for measuring microbial soil in order to gauge the cleaning effect and infection risk for patients.

Current cleaning specifications may not be targeting the correct sites, or, if they are, they may not be applied frequently enough. Cleaning and disinfection should be focused on routine decontamination of high-risk surfaces, i.e., the sites more likely to harbor pathogens and thus facilitating transmission (43, 237, 239). Removing pathogens from handles, switches, buttons, knobs, and other frequently touched (and often forgotten) sites is more likely to have an impact on patient transmission than cleaning inaccessible surfaces such as high shelves, ledges, or ceilings or low-touch surfaces such as walls and window panes (130). Thoughtful construction of a specification to prioritize the highest-risk sites should also obviate the confusion over who cleans what and how often an item or surface should be cleaned (133, 136, 140, 271). In particular, there is currently no evidence to support the frequency of cleaning a room or bed space while it is occupied by a patient (133, 135). Cleaning specifications should encompass the fact that overall cleaning quality is determined not only by the applied method but also by the appropriateness of the method for the type of surface treated.

CONCLUSION

The importance of clean hospitals has not been widely accepted as a key component in infection control despite the increasing interest in HAI during the latter part of the 20th century (24, 27, 272, 273). Now it is finally receiving the attention it deserves (259, 274). No doubt there will be much more evidence forthcoming over the next few years to support and justify hospital cleaning practices. This is to be welcomed, since it is quite possible that accumulating data on environmental reservoirs and pathogen transmission in health care environments will also benefit healthy people in their homes and the community at large (275). Furthermore, with the advance of antimicrobial resistance increasing for virtually all pathogens, the science underpinning infection control, including cleaning, will attain a status hitherto unrecognized. Preventing the transmission of pathogens will be the main focus of the 21st century unless we rapidly find alternative methods for treating infection other than antimicrobial chemotherapy (274).

REFERENCES

- Dancer SJ. 2009. The role of hospital cleaning in the control of hospital-acquired infection. J. Hosp. Infect. 73:378–385. http://dx.doi.org/10.1016/j.jhin.2009.03.030.
- Dancer SJ. 2008. Importance of the environment in meticillinresistant Staphylococcus aureus acquisition: the case for hospital cleaning. Lancet Infect. Dis. 8:101–113. http://dx.doi.org/10.1016/S1473-3099(07)70241-4.
- Martinez JA, Ruthazer R, Hansjosten K, Barefoot L, Snydman DR. 2003. Role of environmental contamination as a risk factor for acquisition of vancomycin-resistant enterococci in patients treated in a medical intensive care unit. Archives Int. Med. 163:1905–1912. http://dx.doi.org/10.1001/archinte.163.16.1905.
- 4. Tankovic J, Legrand P, de Gatines G, Chemineau V, Brun-Buisson C,

- Duval J. 1994. Characterisation of a hospital outbreak of imipenemresistant *Acinetobacter baumannii* by phenotypic and genotypic typing methods. J. Clin. Microbiol. 32:2677–2681.
- Green J, Wright PA, Gallimore CI, Mitchell O, Morgan-Capner P, Brown DWG. 1998. The role of environmental contamination with small round structured viruses in a hospital outbreak investigated by reverse-transcriptase polymerase chain reaction assay. J. Hosp. Infect. 39:39-45. http://dx.doi.org/10.1016/S0195-6701(98)90241-9.
- Kaatz GW, Gitlin SD, Schaberg DR, Wilson KH, Kauffman CA, Seo SM, Fekety R. 1988. Acquisition of *Clostridium difficile* from the hospital environment. Am. J. Epidemiol. 127:1289–1294.
- Kramer A, Schwebke I, Kampf G. 2006. How long do nosocomial pathogens persist on inanimate surfaces? A systematic review. BMC Infect. Dis. 6:130. http://dx.doi.org/10.1186/1471-2334-6-130.
- Wagenvoort JHT, Sluijsmans W, Penders RJR. 2000. Better environmental survival of outbreak vs. sporadic MRSA isolates. J. Hosp. Infect. 45:231–234. http://dx.doi.org/10.1053/jhin.2000.0757.
- Chiang SR, Chuang YC, Tang HJ, Chen CC, Chen CH, Lee NY, Chou CH, Ko WC. 2009. Intratracheal colistin sulfate for BALB/c mice with early pneumonia caused by carbapenem-resistant *Acinetobacter bau-mannii*. Crit. Care Med. 37:2590–2595. http://dx.doi.org/10.1097/CCM .0b013e3181a0f8e1.
- Lawley TD, Clare S, Deakin LJ, Goulding D, Yen JL, Raisen C, Brandt C, Lovell J, Cooke F, Clark TG, Dougan G. 2010. Use of purified Clostridium difficile spores to facilitate evaluation of health care disinfection regimens. Appl. Environ. Microbiol. 76:6895–6900. http://dx.doi.org/10.1128/AEM.00718-10.
- 11. Kjerulf A, Espersen F, Gutschik E, Majcherczyk PA, Hougen HP, Rygaard J, Høiby N. 1998. Serological diagnosis of experimental *Enterococcus faecalis* endocarditis. APMIS 106:997–1008. http://dx.doi.org/10.1111/j.1699-0463.1998.tb00252.x.
- 12. Teunis PF, Moe CL, Liu P, Miller SE, Lindesmith L, Baric RS, Le Pendu J, Calderon RL. 2008. Norwalk virus: how infectious is it? J. Med. Virol. 80:1468–1476. http://dx.doi.org/10.1002/jmv.21237.
- 13. Eaton KA, Friedman DI, Francis GJ, Tyler JS, Young VB, Haeger J, Abu-Ali G, Whittam TS. 2008. Pathogenesis of renal disease due to enterohemorrhagic *Escherichia coli* in germ-free mice. Infect. Immun. 76:3054–3063. http://dx.doi.org/10.1128/IAI.01626-07.
- Wandall DA, Arpi M, Wandall JH. 1997. A rat model of non-lethal bacterial infection. APMIS 105:187–191. http://dx.doi.org/10.1111/j .1699-0463.1997.tb00557.x.
- 15. Lemmen SW, Hafner H, Zolldan D, Stanzel S, Lutticken R. 2004. Distribution of multi-resistant Gram-negative versus Gram-positive bacteria in the hospital inanimate environment. J. Hosp. Infect. 56:191–197. http://dx.doi.org/10.1016/j.jhin.2003.12.004.
- Bhalla A, Pultz NJ, Gries DM, Ray AJ, Eckstein EC, Aron DC, Donskey CJ. 2004. Acquisition of nosocomial pathogens on hands after contact with environmental surfaces near hospitalised patients. Infect. Control Hosp. Epidemiol. 25:164–167. http://dx.doi.org/10.1086/502369.
- Dancer SJ. 2011. Hospital Cleaning in the 21st Century. Euro. J. Clin. Microbiol. Infect. Dis. 30:1473–1481. http://dx.doi.org/10.1007/s10096 -011-1250-x
- Huang SS, Datta R, Platt R. 2006. Risk of acquiring antibiotic-resistant bacteria from prior room occupants. Arch. Intern. Med. 166:1945–1951. http://dx.doi.org/10.1001/archinte.166.18.1945.
- Drees M, Snydman DR, Schmid CH, Barefoot L, Hansjosten K, Vue PM, Cronin M, Nasraway SA, Golan Y. 2008. Prior environmental contamination increases the risk of acquisition of vancomycin-resistant enterococci. Clin. Infect. Dis. 46:678–685. http://dx.doi.org/10.1086/527394.
- Wilks M, Wilson A, Warwick S, Price E, Kennedy D, Ely A, Millar MR. 2006. Control of an outbreak of multidrug-resistant *Acinetobacter baumanii cal-coaceticus* colonization and infection in an intensive care unit (ICU) without closing the ICU or placing patients in isolation. Infect. Control Hosp. Epidemiol. 27:654–658. http://dx.doi.org/10.1086/507011.
- 21. Nseir S, Blazejewski C, Lubret R, Wallet F, Courcol R, Durocher A. 2011. Risk of acquiring multi-drug-resistant Gram-negative bacilli from prior room occupants in the intensive care unit. Clin. Microbiol. Infect. 17:1201–1208. http://dx.doi.org/10.1111/j.1469-0691.2010.03420.x.
- Shaughnessy MK, Micielli RL, DePestel DD, Arndt J, Strachan CL, Welch KB, Chenoweth CE. 2011. Evaluation of hospital room assignment and acquisition of *Clostridium difficile* infection. Infect. Control Hosp. Epidemiol. 32:201–206. http://dx.doi.org/10.1086/658669.

- Dancer SJ, Carling PC. 2010. All that glistens may be neither gold nor clean. J. Hosp. Infect. 76:177–178. http://dx.doi.org/10.1016/j.jhin.2010 06.008
- Dancer SJ. 1999. Mopping up hospital infection. J. Hosp. Infect. 43:85– 100. http://dx.doi.org/10.1053/jhin.1999.0616.
- Nightingale F. 1863. Notes on hospitals. Published by Longman, Green, Longman, Roberts, and Green, London, United Kingdom. http://archive.org/details/notesonhospital01nighgoog. Accessed September 2014.
- Dharan S, Mourouga P, Copin P, Bessmer G, Tschanz B, Pittet D. 1999. Routine disinfection of patients' environmental surfaces: myth or reality? J. Hosp. Infect. 42:113–117. http://dx.doi.org/10.1053/jhin.1999 0567.
- Dettenkofer M, Wenzler S, Amthor S, Antes G, Motschall E, Daschner FD. 2004. Does disinfection of environmental surfaces influence nosocomial infection rates? A systematic review. Am. J. Infect. Control 32:84–89. http://dx.doi.org/10.1016/j.ajic.2003.07.006.
- 28. Davies S. 2005. Hospital contract cleaning and infection control. UNISON, London, United Kingdom.
- Chan P, Dipper A, Kelsey P, Harrison J. 2010. Newspaper reporting of meticillin-resistant *Staphylococcus aureus* and 'the dirty hospital.' J. Hosp. Infect. 75:318–322. http://dx.doi.org/10.1016/j.jhin.2010.01.027.
- Davies S. 2009. Making the connections: contract cleaning and infection control. UNISON, London, United Kingdom.
- National Patient Safety Agency. 2007. The national specifications for cleanliness in the NHS: a framework for setting and measuring performance outcomes. NPSA, London, United Kingdom.
- Healthcare Commission. 2008. Inspections of cleanliness and infection control: how well are acute trusts following the hygiene code. http://www .official-documents.gov.uk/document/hc0809/hc07/0718/0718.pdf. Accessed June 2014.
- Popp W. 2013. Cleaning—on the way to evidence-based knowledge. Healthcare Infect. 18:1–2. http://dx.doi.org/10.1071/HI13004.
- Harbarth S. 2013. Hospital-based environmental hygiene: priorities for research. Healthcare Infect. 18:49–50. http://dx.doi.org/10.1071 /HI13006.
- 35. Mitchell B, Dancer SJ, Shaban RZ, Graves N. 2013. Moving forward with hospital cleaning. Am. J. Infect. Control 41:1138–1139. http://dx.doi.org/10.1016/j.ajic.2013.03.307.
- Cozad A, Jones RD. 2003. Disinfection and the prevention of infectious disease. Am. J. Infect. Control 31:243–254. http://dx.doi.org/10.1067/mic.2003.49.
- 37. Daschner FD, Schuster A, Dettenkofer M, Kümmerer K. 2004. No routine surface disinfection. Am. J. Infect. Control 32:513–515. http://dx.doi.org/10.1016/j.ajic.2004.07.007.
- 38. Trucano M, Kaldenberg D. 2007. The relationship between patient perceptions of hospital practices and facility infection rates: evidence from Pennsylvania hospitals. Patient Safety and Quality Healthcare. http://www.psqh.com/enews/0807feature.html. Accessed June 2014.
- Greaves F, Pape UJ, King D, Darzi A, Majeed A, Wachter RM, Millett C. 2012. Associations between Web-based patient ratings and objective measures of hospital quality. Arch. Intern. Med. 172:435–436. http://dx.doi.org/10.1001/archinternmed.2011.1675.
- Donskey CJ. 2013. Does improving surface cleaning and disinfection reduce health care-associated infections? Am. J. Infect. Control 41:S12– S19. http://dx.doi.org/10.1016/j.ajic.2012.12.010.
- 41. **Hobday RA, Dancer SJ.** 2013. Historical and current perspectives on the role of sunlight and natural ventilation for controlling infection. J. Hosp. Infect. 84:271–282. http://dx.doi.org/10.1016/j.jhin.2013.04.011.
- White L, Dancer SJ, Robertson C, MacDonald J. 2008. Are hygiene standards useful in assessing infection risk? Am. J. Infect. Control 36: 381–384. http://dx.doi.org/10.1016/j.ajic.2007.10.015.
- 43. Dancer SJ, White LF, Lamb J, Girvan EK, Robertson C. 2009. Measuring the effect of enhanced cleaning in a UK hospital: a prospective cross-over study. BMC Med. 7:28. http://dx.doi.org/10.1186/1741-7015-7-28.
- 44. Orenstein R, Aronhalt KC, McManus JE, Jr, Fedraw LA. 2011. A targeted strategy to wipe out *Clostridium difficile*. Infect. Control Hosp. Epidemiol. 32:1137–1139. http://dx.doi.org/10.1086/662586.
- Salgado CD, Sepkowitz KA, John JF, Cantey JR, Attaway HH, Freeman KD, Sharpe PA, Michels HT, Schmidt MG. 2013. Copper surfaces reduce the rate of healthcare-acquired infections in the intensive care unit. Infect. Control Hosp. Epidemiol. 34:479–486. http://dx.doi.org/10.1086/670207.

- Boyce JM, Potter-Bynoe G, Chenevert C, King T. 1997. Environmental contamination due to methicillin-resistant *Staphylococcus aureus*: possible infection control implications. Infect. Control Hosp. Epidemiol. 18: 622–627. http://dx.doi.org/10.1086/647686.
- 47. Hess AS, Shardell M, Johnson JK, Thom KA, Roghmann MC, Netzer G, Amr S, Morgan DJ, Harris AD. 2013. A randomized controlled trial of enhanced cleaning to reduce contamination of healthcare worker gowns and gloves with multidrug-resistant bacteria. Infect. Control Hosp. Epidemiol. 34:487–493. http://dx.doi.org/10.1086/670205.
- 48. Layton MC, Perez M, Heald P, Patterson JE. 1993. An outbreak of mupirocin-resistant *Staphylococcus aureus* on a dermatology ward associated with an environmental reservoir. Infect. Control Hosp. Epidemiol. 14:369–375. http://dx.doi.org/10.1086/646764.
- 49. Rampling A, Wiseman S, Davis L, Hyett AP, Walbridge AN, Payne GC, Cornaby AJ. 2001. Evidence that hospital hygiene is important in the control of methicillin-resistant *Staphylococcus aureus*. J. Hosp. Infect. 49:109–116. http://dx.doi.org/10.1053/jhin.2001.1013.
- de Lassence A, Hidri N, Timsit JF, Joly-Guillou ML, Thiery G, Boyer A, Lable P, Blivet A, Kalinowski H, Martin Y, Lajonchere JP, Dreyfuss D. 2006. Control and outcome of a large outbreak of colonization and infection with glycopeptide-intermediate *Staphylococcus aureus* in an intensive care unit. Clin. Infect. Dis. 42:170–178. http://dx.doi.org/10 .1086/498898.
- Datta R, Platt R, Yokoe DS, Huang SS. 2011. Environmental cleaning intervention and risk of acquiring multidrug-resistant organisms from prior room occupants. Arch. Int. Med. 171:491–494. http://dx.doi.org /10.1001/archinternmed.2011.64.
- 52. Simmons S, Morgan M, Hopkins T, Helsabeck K, Stachowiak J, Stibich M. 2013. Impact of a multi-hospital intervention utilising screening, hand hygiene education and pulsed xenon ultraviolet (PX-UV) on the rate of hospital associated meticillin resistant *Staphylococcus aureus* infection. J. Infect. Prevent. 14:172–174. http://dx.doi.org/10.1177/1757177413490813.
- 53. Mitchell BG, Digney W, Locket P, Dancer SJ. 2014. Controlling methicillin-resistant *Staphylococcus aureus* (MRSA) in a hospital and the role of hydrogen peroxide decontamination: an interrupted time series analysis. BMJ Open 4:e004522. http://dx.doi.org/10.1136/bmjopen -2013-004522.
- Noble MA, Isaac-Renton JL, Bryce DL. 1998. The toilet as a transmission vector of vancomycin-resistant enterococci. J. Hosp. Infect. 40:237

 241. http://dx.doi.org/10.1016/S0195-6701(98)90141-4.
- Byers KE, Durbin LJ, Simonton BM, Anglim AM, Adal KA, Farr BM. 1998. Disinfection of hospital rooms contaminated with vancomycinresistant *Enterococcus faecium*. Infect. Control Hosp. Epidemiol. 19:261– 264. http://dx.doi.org/10.1086/647806.
- Lankford MG, Collins S, Youngberg L, Rooney DM, Warren JR, Noskin GA. 2006. Assessment of materials commonly utilized in health care: implications for bacterial survival and transmission. Am. J. Infect. Control 34:258–263. http://dx.doi.org/10.1016/j.ajic.2005.10.008.
- 57. Sample ML, Gravel D, Oxley C, Toye B, Garber G, Ramotar K. 2002. An outbreak of vancomycin-resistant enterococci in a hematologyoncology unit: control by patient cohorting and terminal cleaning of the environment. Infect. Control Hosp. Epidemiol. 23:468–470. http://dx.doi.org/10.1086/502088.
- Hayden MK, Bonten MJ, Blom DW, Lyle EA, van de Vijver DA, Weinstein RA. 2006. Reduction in acquisition of vancomycin-resistant Enterococcus after enforcement of routine environmental cleaning measures. Clin. Infect. Dis. 42:1552–1560. http://dx.doi.org/10.1086/503845.
- Rossini FA, Fagnani R, Leichsenring ML, Dantas SR, Cardoso LG, Levy CE, Moretti ML, Trabasso P. 2012. Successful prevention of the transmission of vancomycin-resistant enterococci in a Brazilian public teaching hospital. Rev. Soc. Bras. Med. Trop. 45:184–188. http://dx.doi .org/10.1590/S0037-86822012000200009.
- Yoon YK, Sim HS, Kim JY, Park DW, Sohn JW, Roh KH, Lee SE, Kim MJ. 2009. Epidemiology and control of an outbreak of vancomycin-resistant enterococci in the intensive care units. Yonsei Med. J. 50:637–643. http://dx.doi.org/10.3349/ymj.2009.50.5.637.
- 61. Grabsch EA, Mahony AA, Cameron DR, Martin RD, Heland M, Davey P, Petty M, Xie S, Grayson ML. 2012. Significant reduction in vancomycin-resistant enterococcus colonization and bacteraemia after introduction of a bleach-based cleaning-disinfection programme. J. Hosp. Infect. 82:234–242. http://dx.doi.org/10.1016/j.jhin.2012.08.010.
- 62. Passaretti CL, Otter JA, Reich NG, Myers J, Shepard J, Ross T, Carroll

- KC, Lipsett P, Perl TM. 2013. An evaluation of environmental decontamination with hydrogen peroxide vapor for reducing the risk of patient acquisition of multidrug-resistant organisms. Clin. Infect. Dis. 56:27–35. http://dx.doi.org/10.1093/cid/cis839.
- 63. Wilcox MH, Fawley WN, Wrigglesworth N, Parnell P, Verity P, Freeman J. 2003. Comparison of the effect of detergent versus hypochlorite cleaning on environmental contamination and incidence of *Clostridium difficile* infection. J. Hosp. Infect. 54:109–114. http://dx.doi.org/10.1016/S0195-6701(02)00400-0.
- MacLeod-Glover N, Sadowski C. 2010. Efficacy of cleaning products for Clostridium difficile. Can. Fam. Phys. 56:417–423.
- Doan L, Forrest H, Fakis A, Craig J, Claxton L, Khare M. 2012. Clinical and cost-effectiveness of eight disinfection methods for terminal disinfection of hospital isolation rooms contaminated with *Clostridium diffi*cile 027. J. Hosp. Infect. 82:114–121. http://dx.doi.org/10.1016/j.jhin .2012.06.014.
- McMullen KM, Zack J, Coopersmith CM, Kollef M, Dubberke E, Warren DK. 2007. Use of hypochlorite solution to decrease rates of Clostridium difficile-associated diarrhoea. Infect. Control Hosp. Epidemiol. 28:205–207. http://dx.doi.org/10.1086/511791.
- 67. Hacek DM, Ogle AM, Fisher A, Robicsek A, Peterson LR. 2010. Significant impact of terminal room cleaning with bleach on reducing nosocomial *Clostridium difficile*. Am. J. Infect. Control 38:350–353. http://dx.doi.org/10.1016/j.ajic.2009.11.003.
- Sitzlar B, Deshpande A, Fertelli D, Kundrapu S, Sethi AK, Donskey CJ. 2013. An environmental disinfection odyssey: evaluation of sequential interventions to improve disinfection of Clostridium difficile isolation rooms. Infect. Control Hosp. Epidemiol. 34:459–465. http://dx.doi.org/10.1086/670217.
- 69. Hughes GJ, Nickerson E, Enoch DA, Ahluwalia J, Wilkinson C, Ayers R, Brown NM. 2013. Impact of cleaning and other interventions on the reduction of hospital-acquired *Clostridium difficile* infections in two hospitals in England assessed using a breakpoint model. J. Hosp. Infect. 84:227–234. http://dx.doi.org/10.1016/j.jhin.2012.12.018.
- Dancer SJ, Kirkpatrick P, Corcoran DS, Christison F, Farmer D, Robertson C. 2013. Approaching zero: temporal effects of a restrictive antibiotic policy on hospital-acquired *Clostridium difficile*, ESBLproducing coliforms and MRSA. Int. J. Antimicrob. Agents 41:137–142. http://dx.doi.org/10.1016/j.ijantimicag.2012.10.013.
- Starr JM, Campbell A, Renshaw E, Poxton IR, Gibson GJ. 2009.
 Spatio-temporal stochastic modelling of *Clostridium difficile*. J. Hosp. Infect. 71:49–56. http://dx.doi.org/10.1016/j.jhin.2008.09.013.
- Scerpella EG, Wanger AR, Armitige L, Anderlini P, Ericsson CD. 1995. Nosocomial outbreak caused by a multiresistant clone of *Acinetobacter baumannii*: results of the case–control and molecular epidemiologic investigations. Infect. Control Hosp. Epidemiol. 16:92–97. http://dx.doi.org/10.2307/30140949.
- Chmielarczyk A, Higgins PG, Wojkowska-Mach J, Synowiec E, Zander E, Romaniszyn D, Gosiewski T, Seifert H, Heczko P, Bulanda M. 2012.
 Control of an outbreak of *Acinetobacter baumannii* infections using vapourized hydrogen peroxide. J. Hosp. Infect. 81:239–245. http://dx.doi.org/10.1016/j.jhin.2012.05.010.
- Denton M, Wilcox MH, Parnell P, Green D, Keer V, Hawkey PM, Evans I, Murphy P. 2004. Role of environmental cleaning in controlling an outbreak of *Acinetobacter baumannii* on a neurosurgical intensive care unit. J. Hosp. Infect. 56:106–110. http://dx.doi.org/10.1016/j.jhin.2003 10.017
- 75. Naranjo JD, Navarro JIV, Busselo MS, Ruíz AM, Hernández JMH, Garmendia MPT, López MIU. 2013. Control of a clonal outbreak of multidrug-resistant Acinetobacter baumannii in a hospital of the Basque country after the introduction of environmental cleaning led by the systematic sampling from environmental objects. Interdisciplin. Perspect. Infect. Dis. 2013:1–9. http://www.hindawi.com/journals/ipid/2013/582 831/. Accessed June 2014.
- Neely A, Maley MP, Warden GD. 1999. Computer keyboards as reservoirs for *Acinetobacter baumannii* in a burn hospital. Clin. Infect. Dis. 29:1358–1359. http://dx.doi.org/10.1086/313463.
- 77. Apisarnthanarak A, Pinitchai U, Thongphubeth K, Yuekyen C, Warren DK, Fraser VJ, Thammasat University Pandrug-Resistant Acinetobacter baumannii Control Group. 2008. A multifaceted intervention to reduce pandrug-resistant Acinetobacter baumannii colonization and infection in 3 intensive care units in a Thai tertiary care center: a 3-year study. Clin. Infect. Dis. 47:760–767. http://dx.doi.org/10.1086/591134.

- 78. La Forgia C, Franke J, Hacek DM, Thomson RB, Jr, Robicsek A, Peterson LR. 2010. Management of a multidrug-resistant *Acinetobacter baumannii* outbreak in an intensive care unit using novel environmental disinfection: a 38-month report. Am. J. Infect. Control 38:259–263. http://dx.doi.org/10.1016/j.ajic.2009.07.012.
- 79. Doidge M, Allworth AM, Woods M, Marshall P, Terry M, O'Brien K, Goh HM, George N, Nimmo GR, Schembri MA, Lipman J, Paterson DL. 2010. Control of an outbreak of carbapenem-resistant *Acinetobacter baumannii* in Australia after introduction of environmental cleaning with a commercial oxidizing disinfectant. Infect. Control Hosp. Epidemiol. 31:418-420. http://dx.doi.org/10.1086/651312.
- 80. Manian FA, Griesenauer S, Senkel D, Setzer JM, Doll SA, Perry AM, Wiechens M. 2011. Isolation of *Acinetobacter baumannii* complex and methicillin-resistant *Staphylococcus aureus* from hospital rooms following terminal cleaning and disinfection: can we do better? Infect. Control Hosp. Epidemiol. 32:667–672. http://dx.doi.org/10.1086/660357.
- Strassle P, Thom KA, Johnsonm JK, Leekha S, Lissauer M, Zhu J, Harris AD. 2012. The effect of terminal cleaning on environmental contamination rates of multidrug-resistant *Acinetobacter baumannii*. Am. J. Infect. Control 40:1005–1007. http://dx.doi.org/10.1016/j.ajic.2012.05 .027.
- Joynson DHM. 1978. Bowls and bacteria. J. Hyg. Cambr. 80:423–425. http://dx.doi.org/10.1017/S0022172400024888.
- Hobson RP, MacKenzie FM, Gould IM. 1996. An outbreak of multiplyresistant *Klebsiella pneumoniae* in the Grampian region of Scotland. J. Hosp. Infect. 33:249–262. http://dx.doi.org/10.1016/S0195-6701(96) 90011-0.
- 84. Randrianirina F, Vedy S, Rakotovao D, Ramarokoto CE, Ratsitohaina H, Carod JF, Ratsima E, Morillon M, Talarmin A. 2009. Role of contaminated aspiration tubes in nosocomial outbreak of *Klebsiella pneumoniae* producing SHV-2 and CTX-M-15 extended-spectrum β-lactamases. J. Hosp. Infect. 72:23–29. http://dx.doi.org/10.1016/j.jhin.2009.02.004.
- 85. Starlander G, Melhus A. 2012. Minor outbreak of extended-spectrum β-lactamase-producing *Klebsiella pneumoniae* in an intensive care unit due to a contaminated sink. J. Hosp. Infect. 82:122–124. http://dx.doi.org/10.1016/j.jhin.2012.07.004.
- 86. Khan A, Dancer SJ, Humphreys H. 2012. Priorities in the control of extended-spectrum β-lactamases (ESBLs) in hospitals. J. Hosp. Infect. 82:85–93. http://dx.doi.org/10.1016/j.jhin.2012.06.013.
- 87. Tacconelli E, Cataldo MA, Dancer SJ, De Angelis G, Falcone M, Frank U, Kahlmeter G, Pan A, Petrosillo N, Rodríguez-Baño J, Singh N, Venditti M, Yokoe DS, Cookson B. 2014. ESCMID guidelines for the management of the infection control measures to reduce transmission of multidrug-resistant Gram-negative bacteria in hospitalized patients. Clin. Microbiol. Infect. 20(Suppl 1):S1–S55. http://dx.doi.org/10.1111/1469-0691.12427.
- D'Agata EM, Venkataraman L, DeGirolami P, Samore M. 1999. Molecular epidemiology of ceftazidime-resistant gram-negative bacilli on inanimate surfaces and their role in cross-transmission during nonoutbreak periods. J. Clin. Microbiol. 37:3065–3067.
- 89. Kac G, Podglajen I, Vaupre' S, Colardelle N, Buu-Hof A, Gutmann L. 2004. Molecular epidemiology of extended spectrum betalactamase producing Enterobacteriaceae isolated from environmental and clinical specimens in a cardiac surgery intensive care unit. Infect. Control Hosp. Epidemiol. 10:852–855. http://dx.doi.org/10.1086/502308.
- Lerner A, Adler A, Abu-Hanna J, Meitus I, Navon-Venezia S, Carmeli Y. 2013. Environmental contamination by carbapenemase-resistant enterobacteriaceae. J. Clin. Microbiol. 51:177–181. http://dx.doi.org/10.1128/JCM.01992-12.
- 91. Guet-Revillet H, Le Monnier A, Breton N, Descamps P, Lecuyer H, Alaabouche I, Bureau C, Nassif X, Zahar JR. 2012. Environmental contamination with extended-spectrum beta-lactamases: is there any difference between *Escherichia coli* and *Klebsiella* spp? Am. J. Infect. Control 40:845–848. http://dx.doi.org/10.1016/j.ajic.2011.10.007.
- 92. Judge C, Galvin S, Burke L, Thomas T, Humphreys H, Fitzgerald-Hughes D. 2013. Search and you will find: detecting extended-spectrum β-lactamase-producing *Klebsiella pneumoniae* from a patient's immediate environment. Infect. Control Hosp. Epidemiol. 34:534–536. http://dx.doi.org/10.1086/670206.
- 93. Kotsanas D, Wijesooriya WR, Korman TM, Gillespie EE, Wright L, Snook K, Williams N, Bell JM, Li HY, Stuart RL. 2013. "Down the drain": carbapenem-resistant bacteria in intensive care unit patients and

- handwashing sinks. Med. J. Aust. 198:267–269. http://dx.doi.org/10.5694/mja12.11757.
- 94. Roux D, Aubier B, Cochard H, Quentin R, van der Mee-Marquet N, HAI Prevention Group of the Réseau des Hygiénistes du Centre. 2013. Contaminated sinks in intensive care units: an underestimated source of extended-spectrum beta-lactamase-producing Enterobacteriaceae in the patient environment. J. Hosp. Infect. 85:106–111. http://dx.doi.org/10.1016/i.jhin.2013.07.006.
- 95. Yang D, Zhang Z. 2008. Biofilm-forming *Klebsiella pneumoniae* strains have greater likelihood of producing extended-spectrum β-lactamases. J. Hosp. Infect. 68:369–371. http://dx.doi.org/10.1016/j.jhin.2008.02.001.
- 96. Vergara-López S, Domínguez MC, Conejo MC, Pascual Á Rodríguez-Baño J. 2013. Wastewater drainage system as an occult reservoir in a protracted clonal outbreak due to metallo-β-lactamase-producing Klebsiella oxytoca. Clin. Microbiol. Infect. 19:E490–E498. http://dx.doi.org/10.1111/1469-0691.12288.
- 97. Lowe C, Willey B, O'Shaughnessy A, Lee W, Lum M, Pike K, Larocque C, Dedier H, Dales L, Moore C, McGeer A, Mount Sinai Hospital Infection Control Team. 2012. Outbreak of extended-spectrum β-lactamase-producing *Klebsiella oxytoca* infections associated with contaminated handwashing sinks. Emerg. Infect. Dis. 18:1242–1247. http://dx.doi.org/10.3201/eid1808.111268.
- Goddard S, Muller MP. 2011. The efficacy of infection control interventions in reducing the incidence of extended-spectrum β-lactamase-producing Enterobacteriaceae in the non-outbreak setting: a systematic review. Am. J. Infect. Control 39:599–601. http://dx.doi.org/10.1016/j.aiic.2010.09.018.
- 99. Virgincar N, Iyer S, Stacey A, Maharjan S, Pike R, Perry C, Wyeth J, Woodford N. 2011. *Klebsiella pneumoniae* producing KPC carbapenemase in a district general hospital in the UK. J. Hosp. Infect. 78:293–296. http://dx.doi.org/10.1016/j.jhin.2011.03.016.
- 100. Soulier A, Barbut F, Ollivier JM, Petit JC, Lienhart A. 1995. Decreased transmission of Enterobacteriaceae with extended-spectrum betalactamases in an intensive care unit by nursing reorganisation. J. Hosp. Infect. 31:89–97. http://dx.doi.org/10.1016/0195-6701(95)90163-9.
- Kerr K, Snelling AM. 2009. Pseudomonas aeruginosa: a formidable and ever-present adversary. J. Hosp. Infect. 73:338–344. http://dx.doi.org/10 .1016/j.jhin.2009.04.020.
- 102. Döring G, Jansen S, Noll H, Grupp H, Frank F, Botzenhart K, Magdorf K, Wahn U. 1996. Distribution and transmission of *Pseudomonas aeruginosa* and *Burkholderia cepacia* in a hospital ward. Pediatr. Pulmonol. 21:90–100. http://dx.doi.org/10.1002/(SICI)1099-0496(199602) 21:2<90::AID-PPUL5>3.0.CO;2-T.
- 103. Panagea S, Winstanley C, Walshaw MJ, Ledson MJ, Hart CA. 2005. Environmental contamination with an epidemic strain of *Pseudomonas aeruginosa* in a Liverpool cystic fibrosis centre, and study of its survival on dry surfaces. J. Hosp. Infect. 59:102–107. http://dx.doi.org/10.1016/j.jhin.2004.09.018.
- 104. Tall BD, Williams HN, George KS, Gray RT, Walch M. 1995. Bacterial succession within a biofilm in water supply lines of dental air-water syringes. Can. J. Microbiol. 41:647–654. http://dx.doi.org/10.1139/m95 -088.
- 105. Costerton JW, Cheng K-J, Geesey GG, Ladd TI, Nickel JC, Dasgupta M, Marrie TJ. 1987. Bacterial biofilms in nature and disease. Annu. Rev. Microbiol. 41:435–464. http://dx.doi.org/10.1146/annurev.mi.41.100187.002251.
- 106. Weber DJ, Rutala WA, Blanchet CN, Jordan M, Gergen MF. 1999. Faucet aerators: a source of patient colonisation with *Stenotrophomonas maltophilia*. Am. J. Infect. Control 27:59–63. http://dx.doi.org/10.1016/S0196-6553(99)70077-5.
- 107. Doring G, Ulrich M, Muller W, Bitzer J, Schmidt-Koenig L, Münst L, Grupp H, Wolz C, Stern M, Botzenhart K. 1991. Generation of *Pseudomonas aeruginosa* aerosols during hand-washing from contaminated sink drains, transmission to hands of hospital personnel, and its prevention by use of a new heating device. Zentralbl. Hyg. 191:494–505.
- 108. Hota S, Hirji Z, Stockton K, Lemieux C, Dedier H, Wolfaardt G, Gardam MA. 2009. Outbreak of multi-drug resistant *Pseudomonas aeruginosa* colonisation and infection secondary to imperfect intensive care unit room design. Infect. Control Hosp. Epidemiol. 30:25–33. http://dx.doi.org/10.1086/592700.
- 109. Lucero CA, Cohen AL, Trevino I, Rupp AH, Harris M, Forkan-Kelly S, Noble-Wang J, Jensen B, Shams A, Arduino MJ, LiPuma JJ, Gerber SI, Srinivasan A. 2011. Outbreak of *Burkholderia cepacia* complex

- among ventilated paediatric patients linked to hospital sinks. Am. J. Infect. Control 39:775–778. http://dx.doi.org/10.1016/j.ajic.2010.12.005.
- Rutala WA, Weber DJ. 1997. Inorganic hypochlorite (bleach) use in healthcare facilities. Clin. Microbiol. Rev. 10:597–610.
- 111. Thornley CN, Emslie NA, Sprott TW, Greening GE, Rapana JP. 2011. Recurring norovirus transmission on an airplane. Clin. Infect. Dis. 53: 515–520. http://dx.doi.org/10.1093/cid/cir465.
- 112. Evans MR, Meldrum R, Lane W, Gardner D, Ribeiro CD, Gallimore CI, Westmoreland D. 2002. An outbreak of viral gastroenteritis following environmental contamination at a concert hall. Epidemiol. Infect. 129:355–360.
- 113. Isakbaeva ET, Widdowson MA, Beard RS, Bulens SN, Mullins J, Monroe SS, Bresee J, Sassano P, Cramer EH, Glass RI. 2005. Norovirus transmission on cruise ship. Emerg. Infect. Dis. 11:154–158. http://dx.doi.org/10.3201/eid1101.040434.
- 114. Love SS, Jiang X, Barrett E, Farkas T, Kelly S. 2002. A large hotel outbreak of Norwalk-like virus gastroenteritis among three large groups of guests and hotel employees in Virginia. Epidemiol. Infect. 129:127–132.
- 115. Carling PC, Bruno-Murtha LA, Griffiths JK. 2009. Cruise ship environmental hygiene and the risk of norovirus infection outbreaks: an objective assessment of 56 vessels over 3 years. Clin. Infect. Dis. 49:1312–1317. http://dx.doi.org/10.1086/606058.
- 116. Wu HM, Fornek M, Schwab KJ, Chapin AR, Gibson K, Schwab E, Spencer C, Henning K. 2005. A norovirus outbreak at a long-term-care facility: the role of environmental surface contamination. Infect. Control Hosp. Epidemiol. 26:802–810. http://dx.doi.org/10.1086/502497.
- CDC. 2008. Norovirus outbreak in an elementary school—District of Columbia, February 2007. MMWR Morb. Mortal. Wkly. Rep. 56:1340– 1343.
- 118. Luchs A, Morillo SG, Ribeiro CD, Vilanova BC, Calux SJ, Carmona RC, Timenetsky MST. 2011. Gastroenteritis outbreak due to G2P[4] rotavirus and GII norovirus at two correctional facilities in Brazil, 2010. J. Clin. Virol. 51:213–214. http://dx.doi.org/10.1016/j.jcv.2011.04.006.
- 119. Morter S, Bennet G, Fish J, Richards J, Allen DJ, Nawaz S, Iturriza-Gomara M, Brolly S, Gray J. 2011. Norovirus in the hospital setting: virus introduction and spread within the hospital environment. J. Hosp. Infect. 77:106–112. http://dx.doi.org/10.1016/j.jhin.2010.09.035.
- 120. Barker J, Vipond IB, Bloomfield SF. 2004. Effects of cleaning and disinfection in reducing the spread of norovirus contamination via environmental surfaces. J. Hosp. Infect. 58:42–49. http://dx.doi.org/10.1016/j.jhin.2004.04.021.
- 121. Tuladhar E, Hazeleger WC, Koopmans M, Zwietering MH, Beumer RR, Duizer E. 2012. Residual viral and bacterial contamination of surfaces after cleaning and disinfection. Appl. Environ. Microbiol. 78:7769–7775. http://dx.doi.org/10.1128/AEM.02144-12.
- 122. NHS National Services Scotland. 2009. The NHS Scotland national cleaning services specification. Health Facilities Scotland, Glasgow, Scotland
- 123. Sehulster L, Chinn RY, CDC, HICPAC. 2003. Guidelines for environmental infection control in health-care facilities. Recommendations of CDC and the Healthcare Infection Control Practices Advisory Committee (HICPAC). MMWR Recomm. Rep. 52:1–42.
- 124. National Institute of Health and Clinical Excellence. 2011. PH36. Prevention and control of healthcare-associated infections: guidance. http://www.nice.org.uk/nicemedia/live/13763/59578/59578.pdf. Accessed April 2014.
- 125. White L, Dancer SJ, Robertson C. 2007. A microbiological evaluation of hospital cleaning methods. Int. J. Environ. Health Res. 17:285–295. http://dx.doi.org/10.1080/09603120701372433.
- 126. Hegstad K, Langsrud S, Lunestad BT, Scheie AA, Sunde M, Yazdankhah SP. 2010. Does the wide use of quaternary ammonium compounds enhance the selection and spread of antimicrobial resistance and thus threaten our health? Microb. Drug Resist. 16:91–104. http://dx.doi.org/10.1089/mdr.2009.0120.
- 127. Bridier A, Briandet R, Thomas V, Dubois-Brissonnet F. 2011. Comparative biocidal activity of peracetic acid, benzalkonium chloride and ortho-phthalaldehyde on 77 bacterial strains. J. Hosp. Infect. 78:208–213. http://dx.doi.org/10.1016/j.jhin.2011.03.014.
- 128. Vickery K, Deva A, Jacombs A, Allan J, Valente P, Gosbell IB. 2012. Presence of biofilm containing viable multiresistant organisms despite terminal cleaning on clinical surfaces in an intensive care unit. J. Hosp. Infect. 80:52–55. http://dx.doi.org/10.1016/j.jhin.2011.07.007.

- 129. Dancer SJ. 2004. How do we assess hospital cleaning? A proposal for microbiological standards for surface hygiene in hospitals. J. Hosp. Infect. 56:10–15. http://dx.doi.org/10.1016/j.jhin.2003.09.017.
- 130. Smith SJ, Young V, Robertson C, Dancer SJ. 2012. Cross-transmission audit of environmental surfaces, clinical equipment and patient: who touches what? J. Hosp. Infect. 80:206–211. http://dx.doi.org/10.1016/j.jhin.2011.12.007.
- 131. Oelberg DG, Joyner SE, Jiang X, Laborde D, Islam MP, Pickering LK. 2000. Detection of pathogen transmission in neonatal nurseries using DNA markers as surrogate indicators. Pediatrics 105:311–315. http://dx .doi.org/10.1542/peds.105.2.311.
- 132. Griffith C. 2006. HACCP and the management of healthcare associated infections: are there lessons to be learnt from other industries? Int. J. Health Care Qual. Assur. Inc. Leadersh. Health Serv. 19:351–367.
- Bogusz A, Stewart M, Hunter J, Yip B, Reid D, Robertson C, Dancer SJ. 2013. How quickly do hospital surfaces become contaminated after detergent cleaning? Healthcare Infect. 18:3–9. http://dx.doi.org/10.1071 /HI12063.
- 134. Hardy KJ, Gossain S, Henderson N, Drugan C, Oppenheim BA, Gao F, Hawkey PM. 2007. Rapid recontamination with MRSA of the environment of an intensive care unit after decontamination with hydrogen peroxide vapour. J. Hosp. Infect. 66:360–368. http://dx.doi.org/10.1016/j.jhin.2007.05.009.
- 135. Aldeyab MA, McElnay JC, Elshibly SM, Hughes CM, McDowell DA, McMahon MA, Scott MG, Kearney MP. 2009. Evaluation of the efficacy of a conventional cleaning regimen in removing methicillin-resistant *Staphylococcus aureus* from contaminated surfaces in an intensive care unit. Infect. Control Hosp. Epidemiol. 30:304–306. http://dx.doi.org/10.1086/595964.
- 136. Anderson RE, Young V, Stewart M, Robertson C, Dancer SJ. 2011. Cleanliness audit of clinical surfaces and equipment: who cleans what? J. Hosp. Infect. 78:178–181. http://dx.doi.org/10.1016/j.jhin.2011.01.030.
- Schabrun S, Chipchase L. 2006. Healthcare equipment as a source of nosocomial infection: a systematic review. J. Hosp. Infect. 63:239–245. http://dx.doi.org/10.1016/j.jhin.2005.10.013.
- Zachary KC, Bayne PS, Morrison VJ, Ford DS, Silver LC, Hooper DC. 2001. Contamination of gowns, gloves, and stethoscopes with vancomycin-resistant enterococci. Infect. Control Hosp. Epidemiol. 22:560–564. http://dx.doi.org/10.1086/501952.
- Dancer SJ. 2012. Infection control 'undercover': a patient experience. J. Hosp. Infect. 80:189–191. http://dx.doi.org/10.1016/j.jhin.2011.12.003.
- 140. Dumigan DJ, Boyce JM, Havill NL, Golebiewski M, Balogun O, Rizvani R. 2010. Who is really caring for your environment of care? Developing standardized cleaning procedures and effective monitoring techniques. Am. J. Infect. Control 38:387–392. http://dx.doi.org/10.1016/j.ajic.2009.07.005.
- Olesen EM. 2004. Are you 'too posh to wash'? Nursing 34:57. http://dx.doi.org/10.1097/00152193-200411000-00046.
- 142. Moore G, Griffith C. 2006. A laboratory evaluation of the decontamination properties of microfibre cloths. J. Hosp. Infect. 64:379–385. http://dx.doi.org/10.1016/j.jhin.2006.08.006.
- 143. Smith D, Gillanders S, Holah J, Gush C. 2011. Assessing the efficacy of different microfibre cloths at removing surface microorganisms associated with healthcare-associated infections. J. Hosp. Infect. 78:182–186. http://dx.doi.org/10.1016/j.jhin.2011.02.015.
- 144. Wren MWD, Rollins MSM, Jeanes A, Hall TJ, Coen PG, Gant VA. 2008. Removing bacteria from hospital surfaces: a laboratory comparison of ultra microfiber and standard cloths. J. Hosp. Infect. 70:265–271. http://dx.doi.org/10.1016/j.jhin.2008.07.017.
- 145. Mafu AA, Massicotte R, Pichette G, Lafleur S, Lemay MJ, Ahmad D. 2013. Influence of surface and cloth characteristics on mechanical removal of meticillin-resistant *Staphylococcus aureus* (MRSA) attached to inanimate environmental surfaces in hospital and healthcare facilities. Int. J. Infect. Control 9:3.
- 146. Gant VA, Jeanes A, Hall TJ. 2010. Response to: Griffith CJ, Dancer SJ. Hospital cleaning: problems with steam cleaning and microfibre. J. Hosp. Infect. 74:82–84. http://dx.doi.org/10.1016/j.jhin.2009.09.006.
- 147. Scott E, Bloomfield SF. 1990. The survival and transfer of microbial contamination via cloths, hands and utensils. J. Appl. Bacteriol. 68:271–278. http://dx.doi.org/10.1111/j.1365-2672.1990.tb02574.x.
- 148. Bergen LK, Meyer M, Hog M, Rubenhagen B, Andersen LP. 2009. Spread of bacteria on surfaces when cleaning with microfibre cloths. J. Hosp. Infect. 71:132–137. http://dx.doi.org/10.1016/j.jhin.2008.10.025.

- 149. Gibson KE, Crandall PG, Ricke SC. 2012. Removal and transfer of viruses on food contact surfaces by cleaning cloths. Appl. Environ. Microbiol. 78:3037–3044. http://dx.doi.org/10.1128/AEM.00027-12.
- 150. Engelhart S, Krizek L, Glasmacher A, Fischnaller E, Marklein G, Exner M. 2002. *Pseudomonas aeruginosa* outbreak in a haematology-oncology unit associated with contaminated surface cleaning equipment. J. Hosp. Infect. 52:93–98. http://dx.doi.org/10.1053/jhin.2002.1279.
- 151. Forder AA. 1973. Buckets and mops in operating theatres. Lancet i:1325.
- Wishart MM, Riley TV. 1976. Infection with Pseudomonas maltophilia: hospital outbreak due to contaminated disinfectant. Med. J. Aust. 2:710–712.
- 153. McAllister TA, Lucas CE, Mocan H, Liddell RH, Gibson BE, Hann IM, Platt DJ. 1989. *Serratia marcescens* outbreak in a paediatric oncology unit traced to contaminated chlorhexidine. Scott. Med. J. 34:525–528.
- 154. Naparstek L, Carmeli Y, Chmelnitsky I, Banin E, Navon-Venezia S. 2012. Reduced susceptibility to chlorhexidine among extremely-drugresistant strains of *Klebsiella pneumoniae*. J. Hosp. Infect. 81:15–19. http://dx.doi.org/10.1016/j.jhin.2012.02.007.
- 155. Werry C, Lawrence JM, Sanderson PJ. 1988. Contamination of detergent cleaning solutions during hospital cleaning. J. Hosp. Infect. 11:44–49. http://dx.doi.org/10.1016/0195-6701(88)90038-2.
- 156. Reed CS, Barrett SP, Threlfall EJ, Cheasty T. 1995. Control of infection with multiple antibiotic resistant bacteria in a hospital renal unit: the value of plasmid characterisation. Epidemiol. Infect. 115:61–70. http://dx.doi.org/10.1017/S095026880005812X.
- 157. Shlaes DM, Currie-McCumber C, Eanes M, Rotter G, Floyd R. 1986. Gentamicin-resistance plasmids in an intensive care unit. Infect. Control 7:355–361.
- 158. Warnes SL, Highmore CJ, Keevil CW. 2012. Horizontal transfer of antibiotic resistance genes on abiotic touch surfaces: implications for public health. mBio 3(6):e00489-12. http://dx.doi.org/10.1128/mBio .00489-12.
- 159. Chagas TPG, Seki LM, da Silva DM, Asensi MD. 2011. Occurrence of KPC-2-producing Klebsiella pneumoniae strains in hospital wastewater. J. Hosp. Infect. 77:281–282. http://dx.doi.org/10.1016/j.jhin.2010.10.008.
- 160. Gillespie EE, Scott C, Wilson J, Stuart R. 2012. Pilot study to measure cleaning effectiveness in healthcare. Am. J. Infect. Control **40**:477–478. http://dx.doi.org/10.1016/j.ajic.2011.06.010.
- Gillespie E, Wilson J, Lovegrove A, Scott C, Abernethy M, Kotsanas D, Stuart R. 2013. Environment cleaning without chemicals in clinical settings. Am. J. Infect. Control 41:461–463. http://dx.doi.org/10.1016/j.ajic .2012.07.003.
- 162. Berendt AE, Turnbull L, Spady D, Rennie R, Forgie SE. 2011. Three swipes and you're out: how many swipes are needed to decontaminate plastic with disposable wipes? Am. J. Infect. Control 39:442–443. http://dx.doi.org/10.1016/j.ajic.2010.08.014.
- 163. Rutala WA, Gergen MF, Weber DJ. 2012. Efficacy of different cleaning and disinfection methods against *Clostridium difficile* spores: importance of physical removal versus sporicidal inactivation. Infect. Control Hosp. Epidemiol. 33:1255–1258. http://dx.doi.org/10.1086/668434.
- 164. Petti S, Polimeni A, Dancer SJ. 2013. Effect of disposable barriers, disinfection and cleaning on controlling methicillin-resistant *Staphylococcus aureus* environmental contamination. Am. J. Infect. Control 41: 836–840. http://dx.doi.org/10.1016/j.ajic.2012.09.021.
- 165. Finegold SM, Sweeney EE, Gaylor DW, Brady D, Miller LG. 1962. Hospital floor decontamination: controlled blind studies in evaluation of germicides. Antimicrob. Agents Chemother. 1962:250–258.
- 166. Finegold SM, Sweeney EE, Miller LG, Silten J, Brady D, Gaylor DW. 1963. Controlled evaluation of environmental decontamination of general surgical wards. Antimicrob. Agents Chemother. (Bethesda) 161: 774–784.
- 167. Sattar S. 2010. Promises and pitfalls of recent advances in chemical means of preventing the spread of nosocomial infections by environmental surfaces. Am. J. Infect. Control 38:S34–S40. http://dx.doi.org/10 .1016/j.ajic.2010.04.207.
- 168. **Russell AD.** 2004. Bacterial adaptation and resistance to antiseptics, disinfectants and preservatives is not a new phenomenon. J. Hosp. Infect. 57:97–104. http://dx.doi.org/10.1016/j.jhin.2004.01.004.
- 169. Sattar SA, Jean-Yves Maillard J-Y. 2013. The crucial role of wiping in decontamination of high-touch environmental surfaces: review of current status and directions for the future. Am. J. Infect. Control 41:S97– S104. http://dx.doi.org/10.1016/j.ajic.2012.10.032.

- 170. Carling PC, Parry MF, Von Beheren SM, Healthcare Environmental Hygiene Study Group. 2008. Identifying opportunities to enhance environmental cleaning in 23 acute care hospitals. Infect. Control Hosp. Epidemiol. 29:1–7. http://dx.doi.org/10.1086/524329.
- 171. Davies A, Pottage T, Bennett A, Walker J. 2011. Gaseous and air decontamination technologies for *Clostridium difficile* in the healthcare environment. J. Hosp. Infect. 77:199–203. http://dx.doi.org/10.1016/j.jhin.2010.08.012.
- 172. Maclean M, MacGregor SJ, Anderson JG, Woolsey GA, Coia JE, Hamilton K, Taggart I, Watson SB, Thakker B, Gettinby G. 2010. Environmental decontamination of a hospital isolation room using high-intensity narrow-spectrum light. J. Hosp. Infect. 76:247–251. http://dx.doi.org/10.1016/j.jhin.2010.07.010.
- 173. Rutala WA, Weber DJ. 2011. Are room decontamination units needed to prevent transmission of environmental pathogens? Infect. Control Hosp. Epidemiol. 32:743–747. http://dx.doi.org/10.1086/661226.
- 174. Berrington AW, Pedler SJ. 1998. Investigation of gaseous ozone for MRSA decontamination of hospital side-rooms. J. Hosp. Infect. 40:61–65. http://dx.doi.org/10.1016/S0195-6701(98)90026-3.
- 175. Department of Health. 2007. An integrated approach to hospital cleaning: microfibre cloth and steam cleaning technology. Department of Health, London, United Kingdom.
- 176. Tanner BD. 2009. Reduction in infection risk through treatment of microbially contaminated surfaces with a novel, portable, saturated steam vapor disinfection system. Am. J. Infect. Control 37:20–27. http://dx.doi.org/10.1016/j.ajic.2008.03.008.
- 177. Sexton JD, Tanner BD, Maxwell SL, Gerba CP. 2011. Reduction in the microbial load on high-touch surfaces in hospital rooms by treatment with a portable saturated steam vapor disinfection system. Am. J. Infect. Control 39:655–662. http://dx.doi.org/10.1016/j.ajic.2010.11.009.
- 178. Abernethy M, Gillespie E, Snook K, Stuart RL. 2013. Microfiber and steam for environmental cleaning during an outbreak. Am. J. Infect. Control 41:1134–1135. http://dx.doi.org/10.1016/j.ajic.2013.02.011.
- 179. Griffith CJ, Dancer SJ. 2009. Hospital cleaning: problems with steam cleaning and microfibre. J. Hosp. Infect. 72:360–361. http://dx.doi.org/10.1016/j.jhin.2009.04.009.
- 180. de Boer HE, van Elzelingen-Dekker CM, van Rheenen-Verberg CM, Spanjaard L. 2006. Use of gaseous ozone for eradication of methicillinresistant *Staphylococcus aureus* from the home environment of a colonized hospital employee. Infect. Control Hosp. Epidemiol. 27:1120– 1122. http://dx.doi.org/10.1086/507966.
- 181. Foegeding P. 1985. Ozone inactivation of Bacillus and Clostridium spore populations and the importance of spore coat to resistance. Food Microbiol. 2:123–124. http://dx.doi.org/10.1016/S0740-0020(85)80005-8.
- 182. Cardoso CC, Fiorini JE, Ferriera LR, Gurjao JW, Amaral LA. 2000. Disinfection of hospital laundry using ozone: microbiological evaluation. Infect. Control Hosp. Epidemiol. 21:248. http://dx.doi.org/10.1086/503216.
- 183. Sharma M, Hudson JB. 2008. Ozone gas is an effective and practical antibacterial agent. Am. J. Infect. Control 36:559–563. http://dx.doi.org/10.1016/j.ajic.2007.10.021.
- 184. Moat J, Cargill J, Shone J, Upton M. 2009. Application of a novel decontamination process using gaseous ozone. Can. J. Microbiol. 55: 928–933. http://dx.doi.org/10.1139/W09-046.
- 185. Memarzadeh F, Olmsted RN, Bartley JM. 2010. Applications of ultraviolet germicidal irradiation disinfection in health care facilities: effective adjunct, but not stand-alone technology. Am. J. Infect. Control 38:S13—S24. http://dx.doi.org/10.1016/j.ajic.2010.04.208.
- 186. Nerandzic MM, Cadnum JL, Pultz MJ, Donskey CJ. 2010. Evaluation of an automated ultraviolet radiation device for decontamination of *Clostridium difficile* and other healthcare-associated pathogens in hospital rooms. BMC Infect. Dis. 10:197. http://dx.doi.org/10.1186/1471-2334 -10-197.
- 187. Nerandzic MM, Cadnum JL, Eckart KE, Donskey CJ. 2012. Evaluation of a hand-held far-ultraviolet radiation device for decontamination of *Clostridium difficile* and other healthcare-associated pathogens. BMC Infect. Dis. 12:120. http://dx.doi.org/10.1186/1471-2334-12-120.
- 188. Umezawa K, Asai S, Inokuchi S, Miyachi H. 2012. A comparative study of the bactericidal activity and daily disinfection housekeeping surfaces by a new portable pulsed UV radiation device. Curr. Microbiol. 64:581–587. http://dx.doi.org/10.1007/s00284-012-0110-y.
- 189. Rutala WA, Gergen MF, Weber DJ. 2010. Room decontamination by

- ultraviolet radiation. Infect. Control Hosp. Epidemiol. 31:1025–1029. http://dx.doi.org/10.1086/656244.
- 190. Boyce JM, Havill NL, Moore BA. 2011. Terminal decontamination of patient rooms using an automated mobile UV light unit. Infect. Control Hosp. Epidemiol. 32:737–742. http://dx.doi.org/10.1086/661222.
- 191. Maclean M, MacGregor SJ, Anderson JG, Woolsey GA. 2009. Inactivation of bacterial pathogens following exposure to light from a 405-nm LED array. Appl. Environ. Microbiol. 75:1932–1937. http://dx.doi.org/10.1128/AEM.01892-08.
- 192. Hamblin MR, Viveros J, Yang C, Ahmadi A, Ganz RA, Tolkoff MJ. 2005. *Helicobacter pylori* accumulates photoactive porphyrins and is killed by visible light. Antimicrob. Agents Chemother. 49:2822–2827. http://dx.doi.org/10.1128/AAC.49.7.2822-2827.2005.
- 193. Falagas ME, Thomaidis PC, Kotsantis IK, Sgouros K, Samonis G, Karageorgopoulos DE. 2011. Airborne hydrogen peroxide for disinfection of the hospital environment and infection control: a systematic review. J. Hosp. Infect. 78:171–177. http://dx.doi.org/10.1016/j.jhin.2010.12.006.
- 194. Hall L, Otter JA, Chewins J, Wengenack NL. 2007. Use of hydrogen peroxide vapor for deactivation of *Mycobacterium tuberculosis* in a biological safety cabinet and a room. J. Clin. Microbiol. 45:810–815. http://dx.doi.org/10.1128/JCM.01797-06.
- 195. Bentley K, Dove BK, Parks SR, Walker JT, Bennett AM. 2012. Hydrogen peroxide vapour decontamination of surfaces artificially contaminated with norovirus surrogate feline calicivirus. J. Hosp. Infect. 80:116–121. http://dx.doi.org/10.1016/j.jhin.2011.10.010.
- 196. Boyce JM, Havill NI., Otter JA, McDonald LC, Adams NM, Cooper T, Thompson A, Wiggs L, Killgore G, Tauman A, Noble-Wang J. 2008. Impact of hydrogen peroxide vapor room decontamination on *Clostridium difficile* environmental contamination and transmission in a health-care setting. Infect. Control Hosp. Epidemiol. 29:723–729. http://dx.doi.org/10.1086/589906.
- 197. Dancer SJ. 2013. Floor wars: the battle for 'clean' surfaces. J. Hosp. Infect. 84:339–340. http://dx.doi.org/10.1016/j.jhin.2013.01.015.
- 198. Otter JA, Puchowicz M, Ryan D, Salkeld JA, Cooper TA, Havill NL, Tuozzo K, Boyce JM. 2009. Feasibility of routinely using hydrogen peroxide vapour to decontaminate rooms in a busy United States hospital. Infect. Control Hosp. Epidemiol. 30:574–577. http://dx.doi.org/10 .1086/597544.
- Pottage T, Macken S, Walker JT, Bennett AM. 2012. Meticillinresistant *Staphylococcus aureus* is more resistant to vapourized hydrogen peroxide than commercial *Geobacillus stearothermophilus* biological indicators. J. Hosp. Infect. 80:41–45. http://dx.doi.org/10.1016/j.jhin.2011 .11.001.
- Havill NL, Moore BA, Boyce JM. 2012. Comparison of the microbiological efficacy of hydrogen peroxide vapour and ultraviolet light processes for room decontamination. Infect. Control Hosp. Epidemiol. 33: 507–512. http://dx.doi.org/10.1086/665326.
- Levin J, Riley LS, Parrish C, English D, Ahn S. 2013. The effect of portable pulsed xenon ultraviolet light after terminal cleaning on hospital-associated *Clostridium difficile* infection in a community hospital. Am. J. Infect. Control 41:746–748. http://dx.doi.org/10.1016/j.ajic.2013 02 010
- 202. Haas JP, Menz J, Dusza S, Montecalvo MA. 2014. Implementation and impact of ultraviolet environmental disinfection in an acute care setting. Am. J. Infect. Control 42:586–590. http://dx.doi.org/10.1016/j.ajic.2013.12.013.
- 203. **Po JL, Carling PC.** 2010. The need for additional investigation of room decontamination processes. Infect. Control Hosp. Epidemiol. 31:776–777. http://dx.doi.org/10.1086/653819.
- Boyce JM. 2009. New approaches to decontamination of rooms after patients are discharged. Infect. Control Hosp. Epidemiol. 30:515–517. http://dx.doi.org/10.1086/598999.
- Kingston D, Noble WC. 1964. Tests on self-disinfecting surfaces. J. Hyg. (Lond.) 62:519–531. http://dx.doi.org/10.1017/S0022172400040237.
- Page K, Wilson M, Parkin IP. 2009. Antimicrobial surfaces and their potential in reducing the role of the inanimate environment in the incidence of hospital-acquired infections. J. Mater. Chem. 19:3819–3831. http://dx.doi.org/10.1039/B818698G.
- Noyce JO, Michels HH, Keevil CW. 2006. Potential use of copper surfaces to reduce survival of epidemic meticillin-resistant *Staphylococcus aureus* in the healthcare environment. J. Hosp. Infect. 63:289–297. http://dx.doi.org/10.1016/j.jhin.2005.12.008.

- Lansdown ABG. 2006. Silver in healthcare: an enigma and pathological fascination. Bull. R. Coll. Pathol. 133:36–38.
- 209. Su W, Wei SS, Hu SQ, Tang JX. 2009. Preparation of TiO(2)/Ag colloids with ultraviolet resistance and antibacterial property using short chain polyethylene glycol. J. Hazard. Mater. 172:716–720. http://dx.doi.org/10.1016/j.jhazmat.2009.07.056.
- Boyce JM, Havill NL, Lipka A, Havill H, Rizvani R. 2010. Variations in hospital daily cleaning performance. Infect. Control Hosp. Epidemiol. 31:99–101. http://dx.doi.org/10.1086/649225.
- 211. Park KD, Kim YS, Han DK, Kim YH, Lee EHB, Suh H, Choi KS. 1998. Bacterial adhesion on PEG modified polyurethane surfaces. Biomaterials 19:851–859. http://dx.doi.org/10.1016/S0142-9612(97)00245-7.
- Hauert R. 2003. A review of modified DLC coatings for biological applications. Diam. Rel. Mater. 12:583–589. http://dx.doi.org/10.1016/S0925-9635(03)00081-5.
- Parkin IP, Palgrave RG. 2005. Self-cleaning coatings. J. Mater. Chem. 15:1689–1695. http://dx.doi.org/10.1039/B412803F.
- Cheng G, Zhang Z, Chen SF, Bryers JD, Jiang SY. 2007. Inhibition of bacterial adhesion and biofilm formation on zwitterionic surfaces. Biomaterials 28:4192–4199. http://dx.doi.org/10.1016/j.biomaterials.2007 .05.041.
- Medlin J. 1997. Germ warfare. Environ. Health Perspect. 105:290–292. http://dx.doi.org/10.1289/ehp.97105290.
- Syed AK, Ghosh S, Love NG, Boles BR. Triclosan promotes Staphylococcus aureus nasal colonization. mBio 5(2):01015-13. http://dx.doi.org /10.1128/mBio.01015-13.
- 217. Skovgaard S, Nielsen LN, Larsen MH, Skov RL, Ingmer H, Westh H. 2013. Staphylococcus epidermidis isolated in 1965 are more susceptible to triclosan than current isolates. PLoS One 8:e62197. http://dx.doi.org/10.1371/journal.pone.0062197.
- Sánchez-Prado L, Llompart M, Lores M, Fernández-Alvarez M, García-Jares C, Cela R. 2006. Further research on the photo-SPME of triclosan. Anal. Bioanal. Chem. 384:1548–1557. http://dx.doi.org/10 .1007/s00216-006-0311-y.
- 219. FDA. 2013. Taking closer look at 'antibacterial' soap. U.S. Food and Drug Administration, Washington, DC. http://www.fda.gov/forconsumers/consumerupdates/ucm378393.htm. Accessed June 2014.
- 220. Sütterlin S, Molin Y, Melhus A. 2014. Relatively high prevalence of genetic and phenotypic silver resistance among *Enterobacter cloacae* isolates, poster P1016. ECCMID, Barcelona, Spain. http://eccmid.meetingxpert.net/ECCMID_699/poster_109206/program.aspx/anchor 109206. Accessed June 2014.
- Taylor L, Phillips P, Hastings R. 2009. Reduction of bacterial contamination in a healthcare environment by silver antimicrobial technology. J. Infect. Prevent. 10:6–12. http://dx.doi.org/10.1177/1757177408099083.
- 222. O'Hanlon SJ, Enright MC. 2009. A novel bactericidal fabric coating with potent in vitro activity against meticillin-resistant *Staphylococcus aureus* (MRSA). Int. J. Antimcrob. Agents 33:427–431. http://dx.doi.org/10.1016/j.ijantimicag.2008.10.020.
- 223. Stobie N, Duffy B, Colreavy J, McHale P, Hinder SJ, McCormack DE. 2010. Dual-action hygienic coatings: benefits of hydrophobicity and silver ion release for protection of environmental and clinical surfaces. J. Coll. Interface Sci. 345:286–292. http://dx.doi.org/10.1016/j.jcis.2010.02 009
- 224. Casey AL, Adams D, Karpanen TJ, Lambert PA, Cookson BD, Nightingale P, Miruszenko L, Shillam R, Christian P, Elliott TSJ. 2010. Role of copper in reducing hospital environment contamination. J. Hosp. Infect. 74:72–77. http://dx.doi.org/10.1016/j.jhin.2009.08.018.
- 225. Harbarth S, Matthias Dancer SJ. 2013. The environment and health-care-acquired infections: why accurate reporting of results and evaluation of biological plausibility are important. Infect. Control Hosp. Epidemiol. 34:996–997. http://dx.doi.org/10.1086/671741.
- 226. Chen LK, Liu YL, Hu A, Chang KC, Lin NT, Lai MJ, Tseng CC. 2013. Potential of bacteriophage ΦAB2 as an environmental biocontrol agent for the control of multidrug-resistant *Acinetobacter baumannii*. BMC Microbiol. 13:154. http://dx.doi.org/10.1186/1471-2180-13-154.
- Klibanov AM. 2007. Permanently microbicidal materials coatings. J. Mater. Chem. 17:2479–2482. http://dx.doi.org/10.1039/B702079A.
- Wilson M. 2003. Light-activated antimicrobial coating for the continuous disinfection of surfaces. Infect. Control Hosp. Epidemiol. 24:782

 784. http://dx.doi.org/10.1086/502136.
- Decraene V, Pratten J, Wilson. 2006. Cellulose acetate containing toluidine blue and rose bengal is an effective antimicrobial coating when

- exposed to white light. Appl. Environ. Microbiol. **72**:4436–4439. http://dx.doi.org/10.1128/AEM.02945-05.
- Matsunaga T, Tomoda R, Nakajima T, Nakamura N, Komine T. 1988.
 Continuous-sterilization system that uses photosemiconductor powders. Appl. Environ. Microbiol. 54:1330–1333.
- 231. Chow WL, Tin AS, Lim WW, Lim J, Kurup A, Ling ML, Tan AL, Ong BC. 2013. Efficacy of titanium dioxide compounds in preventing environmental contamination by meticillin resistant *Staphylococcus aureus* (MRSA). Int. J. Infect. Control 9:2.
- 232. Weber DJ, Rutala WA. 2012. Commentary: self-disinfecting surfaces. Infect. Control Hosp. Epidemiol. 33:10–13. http://dx.doi.org/10.1086/663648.
- 233. Airey P, Verran J. 2007. Potential use of copper as a hygienic surface; problems associated with cumulative soiling and cleaning. J. Hosp. Infect. 67:271–277. http://dx.doi.org/10.1016/j.jhin.2007.09.002.
- Schweizer HP. 2001. Triclosan: a widely used biocide and its link to antibiotics. FEMS Microbiol. Lett. 202:1–7. http://dx.doi.org/10.1111/j .1574-6968.2001.tb10772.x.
- 235. Ojeil M, Jermann C, Holah JJ, Denyer SP, Maillard JY. 2013. Evaluation of new *in vitro* efficacy test for antimicrobial surface activity reflecting UK hospital conditions. J. Hosp. Infect. 85:274–281. http://dx.doi.org/10.1016/j.jhin.2013.08.007.
- 236. Malik RE, Cooper RA, Griffith CJ. 2003. Use of audit tools to evaluate the efficacy of cleaning systems in hospitals. Am. J. Infect. Control 31: 181–187. http://dx.doi.org/10.1067/mic.2003.34.
- 237. Dancer SJ, White L, Robertson C. 2008. Monitoring environmental cleanliness on two surgical wards. Int. J. Environ. Hygiene. 18:357–364. http://dx.doi.org/10.1080/09603120802102465.
- 238. Lewis T, Griffith C, Gallo M, Weinbren M. 2008. A modified ATP benchmark for evaluating the cleaning of some hospital environmental surfaces. J. Hosp. Infect. 69:156–163. http://dx.doi.org/10.1016/j.jhin.2008.03.013.
- Dancer SJ, Coyne M, Samavedam S, Kennedy J, Wallace P. 2006. MRSA acquisition in an intensive care unit. Am. J. Infect. Control 34:10–17. http://dx.doi.org/10.1016/j.ajic.2005.08.009.
- 240. Mitchell B, Wilson F, McGregor A, Dancer SJ. 2013. Methods to evaluate environmental cleanliness in healthcare facilities. Healthcare Infect. 18:23–30. http://dx.doi.org/10.1071/HI12047.
- Mulvey D, Redding P, Robertson C, Woodall C, Kingsmore P, Bedwell D, Dancer SJ. 2011. Finding a benchmark for monitoring hospital clean-liness. J. Hosp. Infect. 77:25–30. http://dx.doi.org/10.1016/j.jhin.2010.08 006.
- 242. Schmidt MG, Attaway HH, Sharpe PA, John J, Jr, Sepkowitz KA, Morgan A, Fairey SE, Singh S, Steed LL, Cantey JR, Freeman KD, Michels HT, Salgado CD. 2012. Sustained reduction of microbial burden on common hospital surfaces through introduction of copper. J. Clin. Microbiol. 50: 2217–2223. http://dx.doi.org/10.1128/JCM.01032-12.
- 243. Amodio E, Cannova L, Villafrate MR, Merendino AM, Aprea L, Calamusa G. 2014. Analytical performance issues: comparison of ATP bioluminescence and aerobic bacterial count for evaluating surface cleanliness in an Italian hospital. J. Occup. Environ. Hyg. 11:D23–7. http://dx.doi.org/10.1080/15459624.2013.852281.
- 244. Kay D, Bartram J, Pruss A, Ashbolt N, Wyer MD, Fleisher JM, Fewtrell L, Rogers A, Rees G. 2004. Derivation of numerical values for the World Health Organization guidelines for recreational waters. Water Res. 38:1296–1304. http://dx.doi.org/10.1016/j.watres.2003.11.032.
- 245. Pasquarella C, Pitzurra O, Savino A. 2000. The index of microbial air contamination. J. Hosp. Infect. 46:241–256. http://dx.doi.org/10.1053/jhin.2000.0820.
- 246. Sherlock O, O'Connell N, Creamer E, Humphreys H. 2009. Is it really clean? An evaluation of the efficacy of four methods for determining hospital cleanliness. J. Hosp. Infect. 72:140–146. http://dx.doi.org/10.1016/j.jhin.2009.02.013.
- 247. Boyce JM, Havill NL, Dumigan DG, Golebiewski M, Balogun O, Rizvani R. 2009. Monitoring the effectiveness of hospital cleaning practices by use of an adenosine triphosphate bioluminescence assay. Infect. Control Hosp. Epidemiol. 30:678–684. http://dx.doi.org/10.1086/598243.
- Aiken ZA, Wilson M, Pratten J. 2011. Evaluation of ATP bioluminescence assays for potential use in a hospital setting. Infect. Control Hosp. Epidemiol. 32:507–509. http://dx.doi.org/10.1086/659761.
- 249. Whiteley GS, Derry C, Glasbey T. 2012. The comparative performance of three brands of portable ATP-bioluminometer intended for use in

- hospital infection control. Healthcare Infect. 17:91–97. http://dx.doi.org/10.1071/HI12021.
- 250. Boyce JM, Havill NL, Havill HL, Mangione E, Dumigan DG, Moore BA. 2011. Comparison of fluorescent marker systems with 2 quantitative methods of assessing terminal cleaning practices. Infect. Control Hosp. Epidemiol. 32:1187–1193. http://dx.doi.org/10.1086/662626.
- Malik DJ, Shama G. 2012. Estimating bacterial surface contamination by means of ATP determinations: 20 pence short of a pound. J. Hosp. Infect. 80:354–355. http://dx.doi.org/10.1016/j.jhin.2011.12.021.
- 252. Brown E, Eder AR, Thompson KM. 2010. Do surface and cleaning chemistries interfere with ATP measurement systems for monitoring patient room hygiene? J. Hosp. Infect. 74:193–195. http://dx.doi.org/10.1016/j.jhin.2009.10.006.
- 253. Chadwick C, Oppenheim BA. 1996. Cleaning as a cost-effective method of infection control. Lancet 347:1776. http://dx.doi.org/10.1016/S0140 -6736(96)90858-2.
- 254. Hota B, Blom DW, Lyle EA, Weinstein RA, Hayden MK. 2009. Interventional evaluation of environmental contamination by vancomycin-resistant enterococci: failure of personnel, product, or procedure? J. Hosp. Infect. 71:123–131. http://dx.doi.org/10.1016/j.jhin.2008.10.030.
- Carling PC, Briggs JL, Perkins J, Highlander D. 2006. Improved cleaning of patient rooms using a new targeting method. Clin. Infect. Dis. 42:385–388. http://dx.doi.org/10.1086/499361.
- Carling P. 2013. Methods for assessing the adequacy of practice and improving room disinfection. Am. J. Infect. Control 41(Suppl 5):S20– S25. http://dx.doi.org/10.1016/j.ajic.2013.01.003.
- 257. Goodman ER, Platt R, Bass R, Onderdonk AB, Yokoe DS, Huang SS. 2008. Impact of an environmental cleaning intervention on the presence of methicillin-resistant *Staphylococcus aureus* and vancomycin-resistant enterococci on surfaces in intensive care unit rooms. Infect. Control Hosp. Epidemiol. 29:593–599. http://dx.doi.org/10.1086/588566.
- 258. Kundrapu S, Sunkesula V, Sitzlar BM, Fertelli D, Deshpande A, Donskey CJ. 2014. More cleaning, less screening: evaluation of the time required for monitoring versus performing environmental cleaning. Infect. Control Hosp. Epidemiol. 35:202–204. http://dx.doi.org/10.1086/674852
- Carling PC, Huang SS. 2013. Improving healthcare environmental cleaning and disinfection. Infect. Control Hosp. Epidemiol. 34:507–513. http://dx.doi.org/10.1086/670222.
- 260. Willis C, Morley R, Westbury J, Greenwood M, Pallett A. 2007. Evaluation of ATP bioluminescence swabbing as a monitoring and training tool for effective hospital cleaning. Br. J. Infect. Control 8:17–21. http://dx.doi.org/10.1177/1469044607083604.
- Eckstein BC, Adams DA, Eckstein EC, Rao A, Sethi AK, Yadavalli GK, Donskey CJ. 2007. Reduction of *Clostridium difficile* and vancomycinresistant Enterococcus contamination of environmental surfaces after an intervention to improve cleaning methods. BMC Infect. Dis. 7:61. http://dx.doi.org/10.1186/1471-2334-7-61.
- 262. Guerrero DM, Carling PC, Jury LA, Ponnada S, Nerandzic MM, Donskey CJ. 2013. Beyond the Hawthorne effect: reduction of *Clostridium difficile* environmental contamination through active intervention to improve cleaning practices. Infect. Control Hosp. Epidemiol. 34:524–526. http://dx.doi.org/10.1086/670213.

- 263. Rupp ME, Adler A, Schellen M, Cassling K, Fitzgerald T, Sholtz L, Lyden E, Carling P. 2013. The time spent cleaning a hospital room does not correlate with the thoroughness of cleaning. Infect. Control Hosp. Epidemiol. 34:100–102. http://dx.doi.org/10.1086/668779.
- 264. Munoz-Price LS, Bimbach DJ, Lubarsky DA, Arheart KL, Fajardo-Aquino Y, Rosalsky M, Cleary T, Depascale D, Coro G, Namias N, Carling P. 2012. Decreasing operating room environmental pathogen contamination through improved cleaning practice. Infect. Control Hosp. Epidemiol. 33:897–904. http://dx.doi.org/10.1086/667381.
- 265. Fitzgerald T, Sholtz LA, Marion N, Turner P, Carling PC, Rupp ME. 2012. Maintenance of environmental services cleaning and disinfection in the ICU after a performance improvement project, poster 15-216. Am. J. Infect. Control 40:e159. http://dx.doi.org/10.1016/j.ajic.2012.04.281.
- 266. Clements A, Halton K, Graves N, Pettitt A, Morton A, Looke D, Whitby M. 2008. Overcrowding and understaffing in modern health-care systems: key determinants in meticillin-resistant *Staphylococcus aureus* transmission. Lancet Infect. Dis. 8:427–434. http://dx.doi.org/10.1016/S1473-3099(08)70151-8.
- 267. Donker T, Wallinga J, Grundmann H. 2014. Dispersal of antibiotic-resistant high-risk clones by hospital networks: changing the patient direction can make all the difference. J. Hosp. Infect. 86:34–41. http://dx.doi.org/10.1016/j.jhin.2013.06.021.
- 268. Stewart M, Bogusz A, Hunter J, Devanny I, Yip B, Reid D, Robertson C, Dancer SJ. 2014. Use of electrolysed water to clean near-patient sites: is disinfectant better than detergent?, poster eP269. ECCMID, Barcelona, Spain.
- 269. Thom KA, Johnson JK, Lee MS, Harris AD. 2011. Environmental contamination because of multidrug-resistant *Acinetobacter baumannii* surrounding colonized or infected patients. Am. J. Infect. Control 39: 711–715. http://dx.doi.org/10.1016/j.ajic.2010.09.005.
- 270. Dancer SJ, Humphreys H, Carling P. 2011. Disinfection is not the same as cleaning. Crit. Care Med. 39:1853–1854. http://dx.doi.org/10.1097/CCM.0b013e31821856de.
- Ptak J, Tostenson L, Kirkland K, Taylor E. 2009. Who is responsible for cleaning that? Am. J. Infect. Control 37:E133–E134. http://dx.doi.org/10 .1016/j.ajic.2009.04.180.
- 272. Haley RW, Quade D, Freeman HE, Bennett JV. 1980. Study on the efficacy of nosocomial infection control (SENIC Project). Summary of study design. Am. J. Epidemiol. 111:472–485.
- 273. Maki DG, Alvarado CJ, Hassemer CA, Zilz MA. 1982. Relation of the inanimate hospital environment to endemic nosocomial infection. N. Engl. J. Med. 307:1562–1566. http://dx.doi.org/10.1056/NEJM19821216 3072507.
- 274. Dancer SJ. 2013. Infection control in the post-antibiotic era. Healthcare Infect. 18:51–60. http://dx.doi.org/10.1071/HI12042.
- 275. Scott E. 2013. Community-based infections and the potential role of common touch surfaces as vectors for the transmission of infectious agents in home and community settings. Am. J. Infect. Control 41:1087–1092. http://dx.doi.org/10.1016/j.ajic.2013.05.014.
- 276. Stewart M, Bogusz A, Hunter J, Devanny I, Yip B, Reid D, Robertson C, Dancer SJ. Use of electrolysed water to clean near-patient sites: is disinfectant better than detergent? Infect. Control Hosp. Epidemiol., in press.

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